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AMERICAN VETERINARY MEDICAL ASSOCIATION

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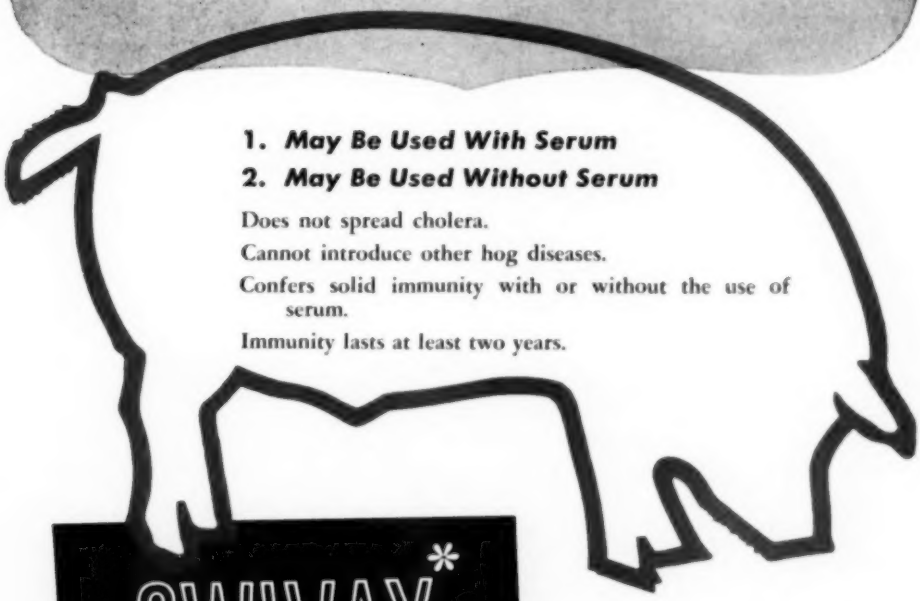

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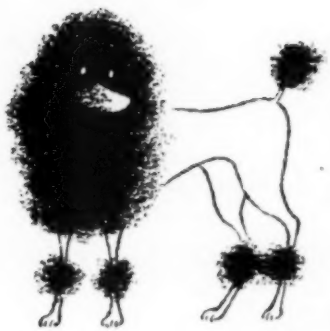

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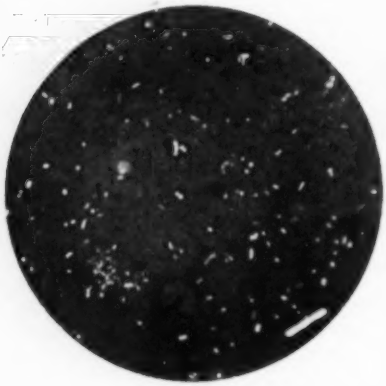

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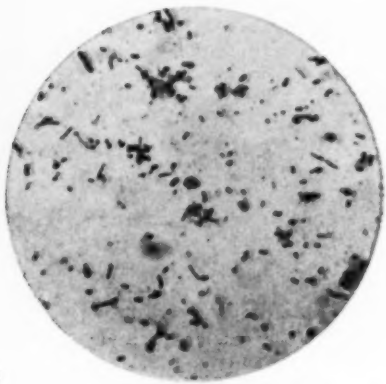

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AVMA ☆ Report

Veterinary Medical Activities

✦ The Board of Governors (Drs. E. Laitinen, chairman, J. A. McCallam, and A. H. Quin) were in session at Association headquarters on September 28 and 29.

★ ★ ★

✦ President J. A. McCallam attended and took part in the programs of the annual meeting of the Pennsylvania State V. M. A. in Pittsburgh, October 14 to 16, and of the New England V. M. A. in Newport, R. I., October 18 to 20.

★ ★ ★

✦ President-Elect A. H. Quin appeared on the "Dinner Bell" radio program of station WLS on September 29 while in Chicago for the meeting of the Board of Governors.

★ ★ ★

✦ Executive Secretary J. G. Hardenbergh attended the 41st annual short course for veterinarians at Purdue University, October 7 to 9.

★ ★ ★

✦ Assistant Executive Secretary, H. E. Kingman, Jr., and Mr. Russell Rongren, in charge of the AVMA public information program, conferred with staff members of Michigan State College at East Lansing on October 1 with respect to the kinescope project which the Association is studying. On the same trip Dr. Kingman attended a committee meeting of the National Conference on Trichinosis in Ann Arbor on October 2.

★ ★ ★

✦ Dr. W. T. S. Thorp, AVMA representative to the National Society for Medical Research, attended a meeting of the board of directors of the Society in Atlantic City on October 27.

★ ★ ★

✦ Dr. J. L. McAuliff, chairman of the AVMA Special Committee on Brucellosis, attended a meeting in Washington, D. C., September 28 and 29, to consider proposed changes in interstate regulations pertaining to brucellosis.

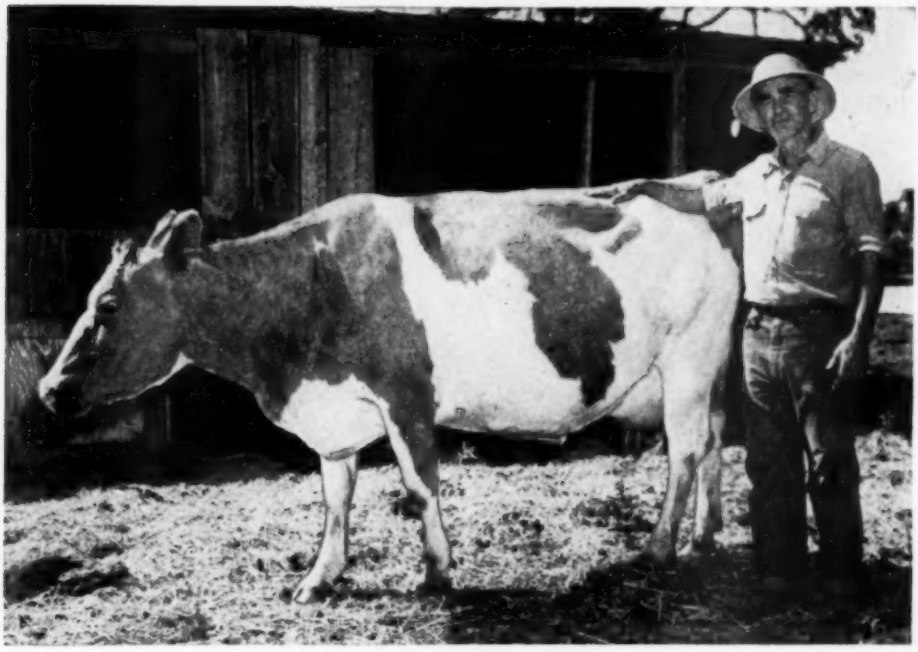
★ ★ ★

✦ Mr. Norman Kraeft, radio farm director of Chicago radio station WGN, plans to distribute tape recordings of 27 interviews with veterinarians at the Toronto meeting to 15 clear channel stations located throughout the United States.

★ ★ ★

✦ Following are some of the meetings of AVMA bodies which will be held at Association headquarters in Chicago in November and December:

November 9 and 10 — Council on Education
November 29 — Research Council
December 1 — Board of Governors
December 2, 3, and 4 — Executive Board



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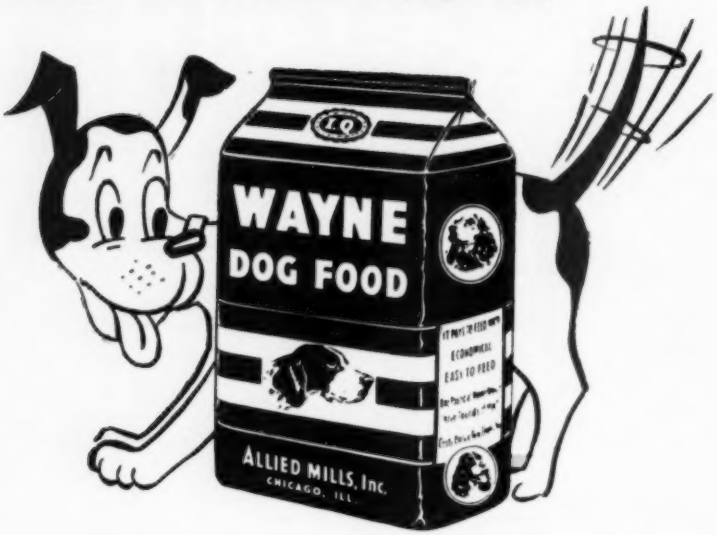

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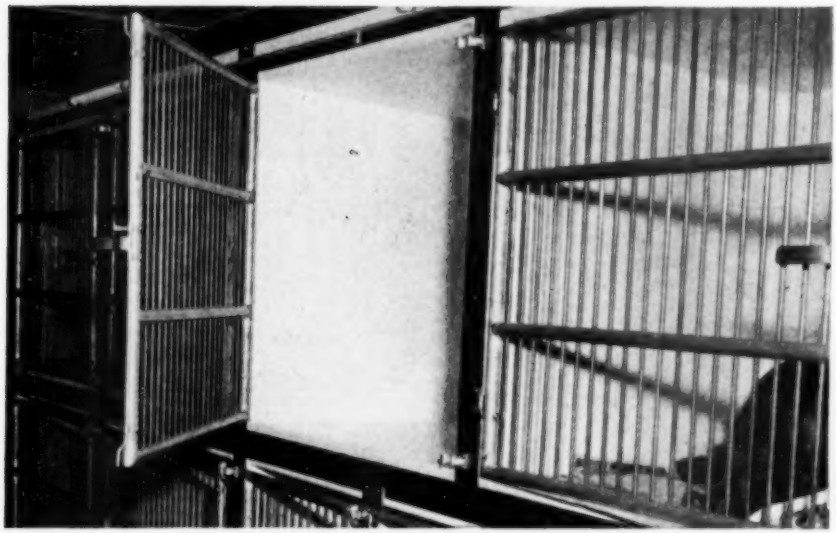

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(1) Whitney, G. D.: J. Am. Vet. M. A. *122*:21, 1953.
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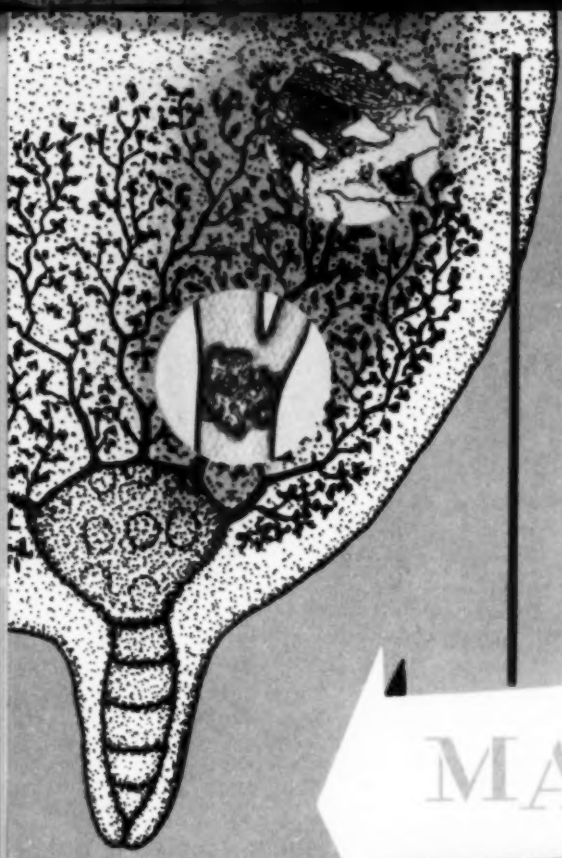

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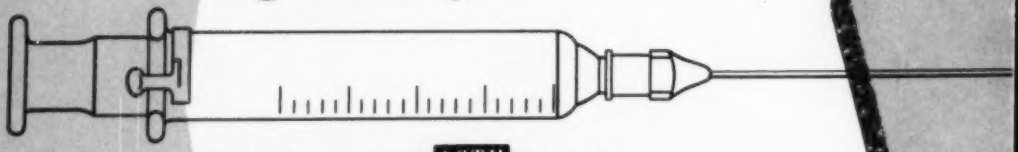

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1. Shaw, J. C., et al.: The North American Veterinarian, 251-256, April, 1953.

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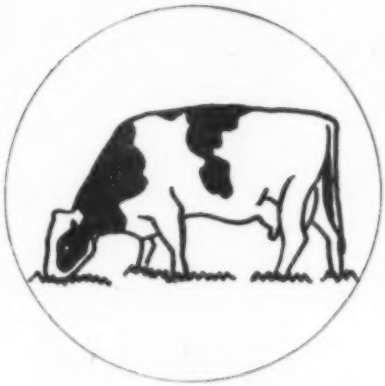

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*Christian, A.B., Segard, C.P.: The Cause and Treatment of Ketosis in Cattle, J.A.V.M.A., 122, (June, 1953): 479-480.

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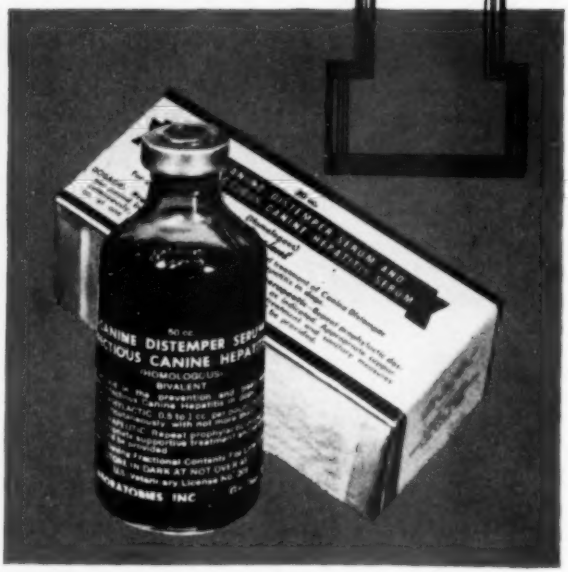

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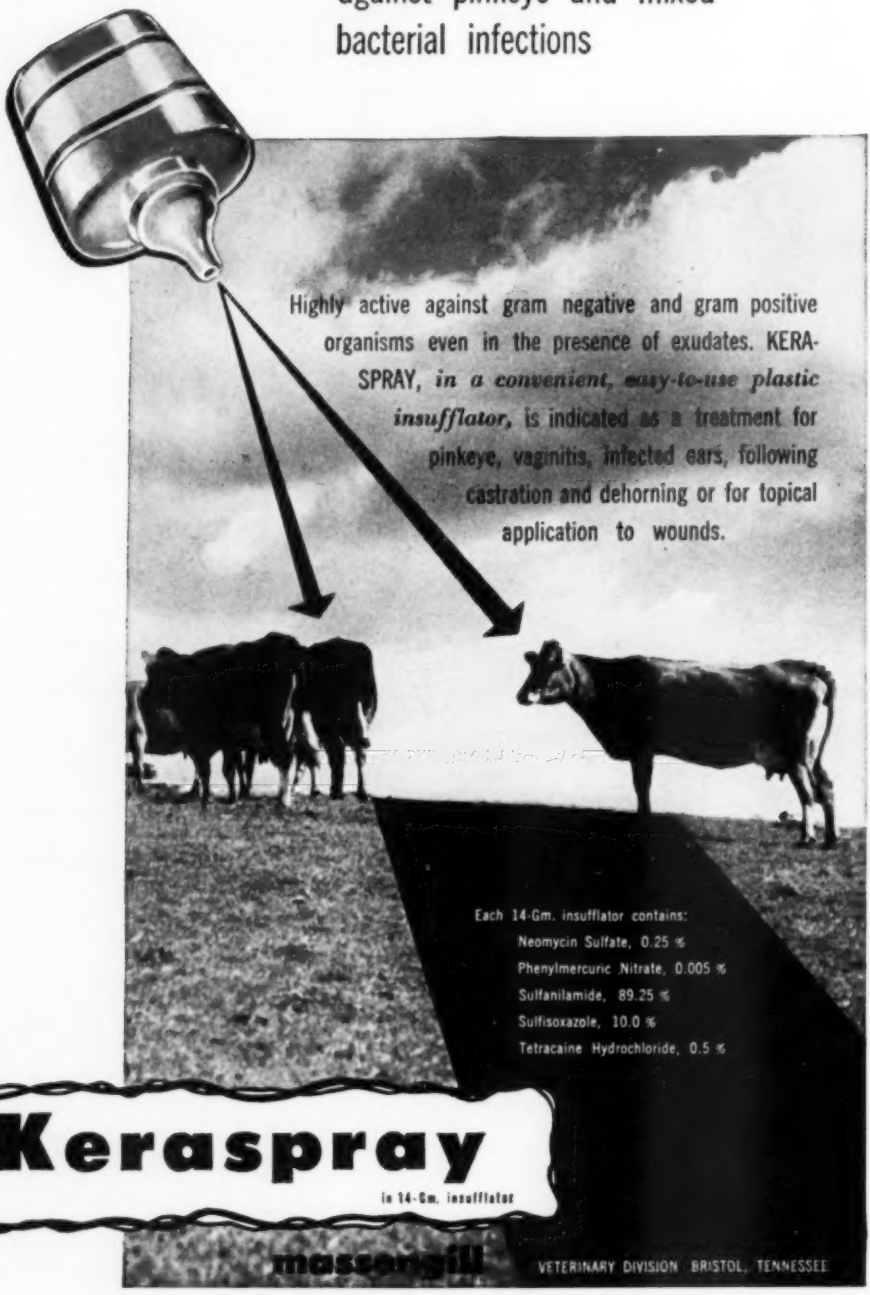
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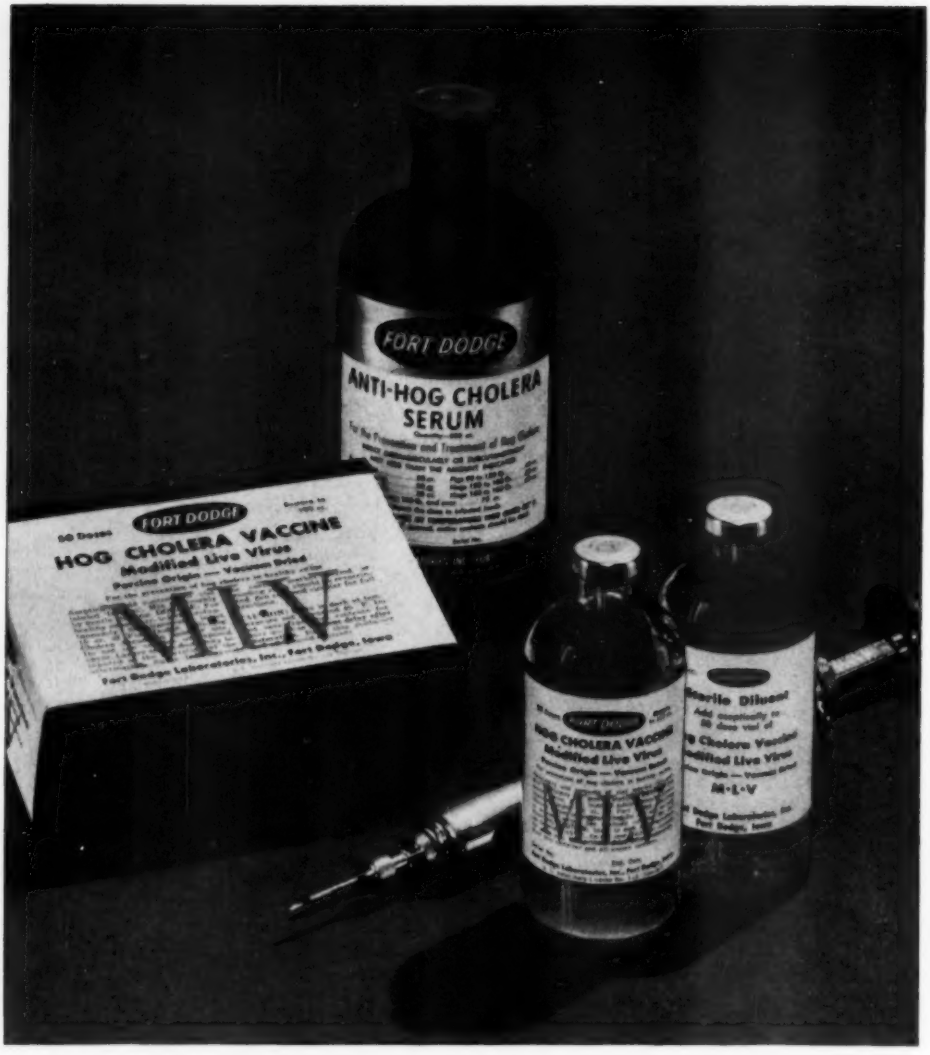

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VOL. 123 NOVEMBER, 1953 No. 920

The Treatment of Chronic Lead Poisoning in Horses with Calcium Disodium Ethylenediaminetetraacetate

L. W. HOLM, Ph.D.; J. D. WHEAT, D.V.M.; E. A. RHODE, D.V.M.; GLADYS FIRCH, B.A.

Davis, California

PAST INVESTIGATIONS in areas adjacent to lead mines, smelters, and refuse dumps have shown that chronic lead intoxication of horses may be of frequent occurrence. Safety measures have largely obviated the extensive lead hazard, but poisoning does occur sporadically and usually manifests itself as chronic poisoning.

The diagnosis of chronic lead poisoning in domestic animals is more difficult than in human patients. The appearance of basophilic stippling of the erythrocytes, the lead line on the gums, and low grade anemia are unpredictable. In horses, the most diagnostic findings are the occurrence of dyspnea with roaring in terminal cases, harsh coats, muscular weakness, and stiffness of the joints sufficient at times to prevent full extension of the members.[12,13] Paralysis of the lips and variations of tonicity of the gut have been reported, but they are not of constant occurrence. Edema of nephritic origin has occurred in some cases.

LITERATURE

Literature concerning exposure concentrations, times of exposure, and tissue levels of lead in normal and in lead-poisoned horses is not as complete as that for other species. No data are available which relate toxicity to the solubility of the various lead compounds. It is said that 500 to 750 Gm. of lead acetate has produced acute signs in horses and that a single dose of 360 Gm. of lead oxide has been sufficient to kill a horse.[4]

Data relative to lead intake in chronic poisoning are more complete. Thomassen (quoted by Fröhner) gave 5 to 20 Gm. of lead carbonate per day to each of 2 foals. Within four to six weeks the animals showed signs of poisoning; emaciation and roaring were the predominant signs. Gabel[5] fed 320 mg. of lead per day to each of 2 horses. A total of 60 Gm. was consumed by each in the course of the trial. One animal showed signs of colic, stiffness, and roughened coat within three weeks. The second animal showed no signs of poisoning until roaring was observed three and a half months after feeding began. Mosselman[16] reported that horses died after being pastured on grass adjacent to a lead works. Here, the foliage had a lead content of 6.5 Gm. per quadrameter.

It is well known that lead is a ubiquitous substance and that all animal tissues contain a small but variable amount of the metal. Lead levels in unexposed horses have been reported by Dankwortt and Höll.[3] Soft tissues such as spleen, kidney, and liver contained 0.01 to 0.025 mg./100 Gm. of tissue. Bone contained 0.33 to 1.20 mg./100 Gm. of tissue. Limited data from this laboratory sets the tissue lead level for this area much lower than the above figures. Horse liver and kidney cortex have contained 0.23 and 0.13 μg./100 Gm. of tissue. Lead levels of defatted bone have been approximately 73.0 μ (0.073 mg.) per 100 Gm.

Lead levels of tissues of horses during chronic poisoning were determined by Gabel.[5] In the 2 horses mentioned above, he found 5.0 to 9.8 mg. of lead per 100 Gm. of liver. However, in 126 cases of chronic lead poisoning brought to autopsy, only 5 had a liver lead concentration greater than 1.0 mg./100 Gm. The average concentration was 0.55 mg./100 Gm. Gregg[10] found a comparable lead level in the liver in one case reported.

From the School of Veterinary Medicine, University of California, Davis.

THERAPY OF LEAD INTOXICATION

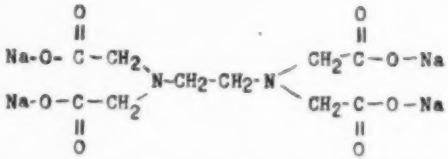
Therapy of lead intoxication in domestic animals has tended to follow classical lines which will be described below. With the advent of the use of chelating agents in medicine, the mode of attack in certain metallic poisonings may well be modified. The chelating agent of direct concern here is ethylenediaminetetraäcetic acid (EDTA) and its tetra sodium salt. EDTA is a versatile and powerful chelating agent, commercially known as versene[36] and sequestrene,[36] which forms soluble, nonionizable complexes with certain polyvalent ions. The tetra sodium salt effectively removes calcium ions from solution. Under alkaline conditions, calcium may be removed from relatively insoluble oxalates. This salt has been used as an anticoagulant for blood *in vitro*.[17]

The calcium disodium salt (Ca EDTA)* complexes with lead in such a manner that lead replaces calcium in the molecule and the latter is freed. The Pb EDTA is nonionizable and is readily excreted. These reactions are shown in the equations in figure 1.

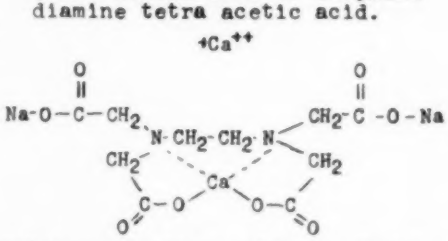
Extensive toxicity trials with Ca EDTA have demonstrated that it is remarkably inert when given to small laboratory animals.[14] Because of the potentialities with respect to lead, Ca EDTA has been tried as a therapeutic agent in several cases of acute lead poisoning in human patients.[15] The above factors suggested the use of this compound in the cases of chronic lead poisoning in horses described below.

MATERIALS AND METHODS

Of 3 geldings presented to the University (of California) clinic on Oct. 25, 1952, 2 were 6-year-olds and 1 was 8 years old. Each weighed approximately 1,000 lb. Prior to admission, they had been quartered on native grass pasture 2 miles from a smelter and within the smoke zone of the smelter. The client stated that several horses had died previously after being pastured in this area. Three liver specimens from the dead animals were submitted for toxicological examination. The clinic patients had harsh coats, ate sparingly, tired easily during exercise, and exhibited moderate to severe dyspnea. Animal 1 was edematous in the belly and legs. Animals 2 and 3 had pharyngeal paralysis and were roarers. In addition, animal 3 had a lead line on the gums and exhibited pneumonic signs. Urine and blood samples were taken from all animals for toxicological analysis. Forty-eight hours after admission, animal 3 died. At autopsy, samples of bone, liver, and kidney were taken for analysis.



Tetra sodium salt of ethylene diamine tetra acetic acid.



Calcium disodium salt of ethylene diamine tetra acetic acid.

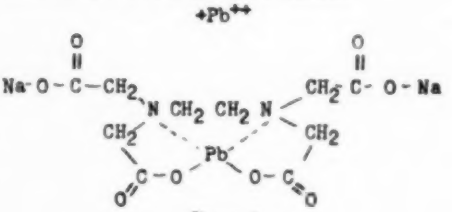


Figure 1

From the preliminary analyses and examination, a diagnosis of chronic lead poisoning was made. Animals 1 and 2 were placed on an intravenous drip of Ca EDTA. The dosage was 1 Gm./30 lb. of body weight at a concentration of 1 Gm./50 cc. of solution. To that end, 132 cc. of a 25 per cent solution of Ca EDTA was diluted to 1,650 cc. with saline and was dripped into the jugular vein at a drop rate of 60 to 80 per minute. Animal 1 was treated daily for three days; animal 2 was treated daily for four days. In the case of animal 1, a four-day lapse occurred before a three-day drip schedule was repeated. In the case of animal 2, three days elapsed between treatments. Blood and urine samples were taken at the same time each day and fecal samples were taken at less frequent intervals.

On November 3, 4 more animals were presented from the same farm. These were 7-year-olds weighing approximately 1,000 lb. each. Two animals (4 and 5) presented the same clinical picture as was seen in the previous cases, but the signs

*Calcium disodium ethylenediaminetetraacetate was supplied to us through the courtesy of the Bersworth Chemical Company, Framingham, Mass., and Riker Laboratories, Inc., Los Angeles, Calif.

were not as marked. Animal 4 was markedly hyperesthetic in the region of the neck and pharynx. On the basis of blood and urine analyses for lead, animals 4 and 5 were placed on a drip of Ca EDTA. The concentration and amount of therapeutic agent was the same as for the first cases but the interval between treatment schedules was increased to five days. Blood, urine, and fecal samples were taken routinely.

In order to determine the lead intake of the affected animals, grass samples of the suspected pasture were taken from four scattered but representative spots. Lead in the tissues and urine was analyzed by the dithizon method as described by the Association of Official Agricultural Chemists.

RESULTS

An autopsy of animal 3 showed that death was caused by gangrenous pneumonia. There was marked portal cirrhosis with biliary hyperplasia. About 700 cc. of dark red fluid was found in the pleural cavity and 300 cc. of a similar fluid was found in the pericardial sac. The lead content of the tissues of this animal was as follows: urine, 0.09 mg./100 cc.; liver, 0.52 mg.; kidney, 0.50 mg.; defatted bone, 1.82 mg./100 Gm., respectively. The level of lead in this liver corresponded well with those levels found in liver specimens taken from the 3 horses previously pastured with the patients. The lead level in the liver of these horses was 0.39, 0.68, and 0.73 mg./100 Gm.

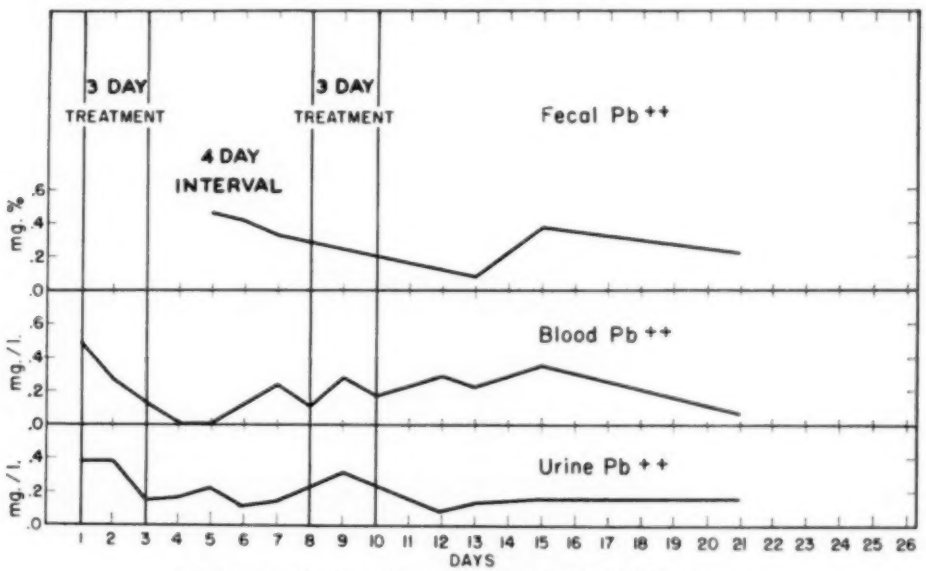
Hematological examination of the poisoned horses showed no abnormalities in the cells, no basophilic stippling, and no anemia.

The lead content of the four samples of pasture grass was 89, 312, 211, and 113 parts per million.

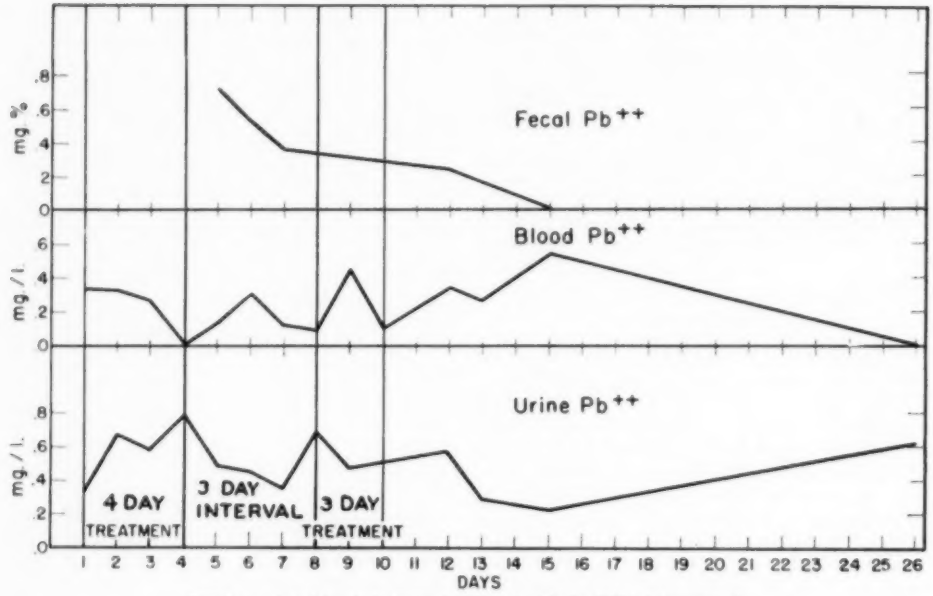
Initial lead concentrations of blood and urine and those found during therapy with Ca EDTA are shown in graphs 1 to 4. No consistent excretion pattern was found.

The sharp urinary excretion peak in animal 5 on day 14 was correlated with known recent exposure of this animal. This animal, therefore, had more lead in the soft tissues than did those animals with a history of long exposure. In the latter animals, depot lead would tend to be high, and Ca EDTA would not mobilize the metal from these sites. Data in this laboratory on excretion of Pb EDTA in acute lead poisoning supports this.

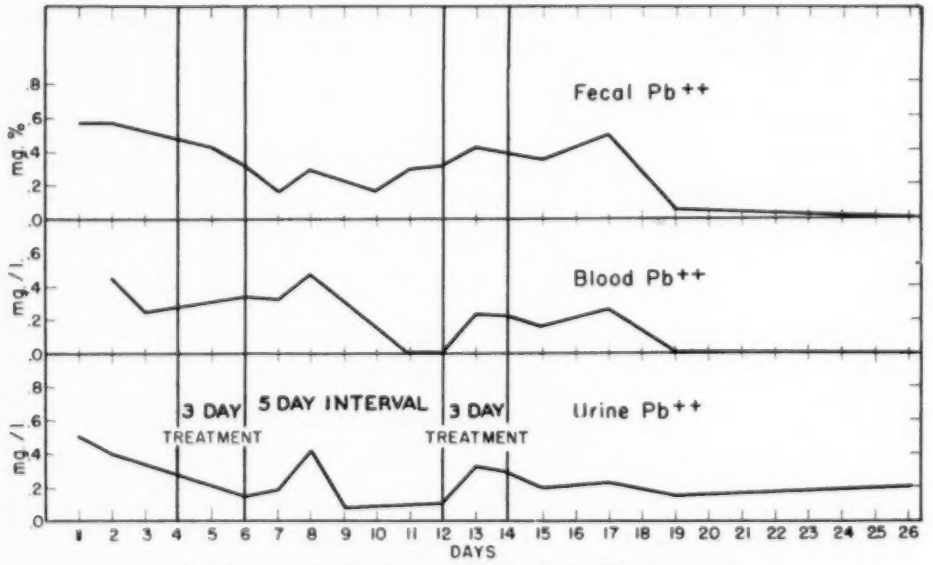
Except for animal 1, all horses showed marked clinical improvement within four days after treatment was initiated. The improvement was especially dramatic in animal 2 which, except for pneumonic signs, was as sick as animal 3 prior to its death. After the first treatment period, the animals became alert and ate well. Respiratory



Graph 1—Lead levels in horse 1, treated with Ca EDTA intravenously.



Graph 2— Lead levels in horse 2, treated with Ca EDTA intravenously.



Graph 3—Lead levels in horse 4, treated with Ca EDTA intravenously.

difficulty did not cease until the end of the second period of treatment. Hyperesthesia of the cervical area in animal 4, possibly caused by incipient vagal paralysis, was not alleviated during treatment. In fact, sensitivity became more pronounced during therapy but returned to normal several days after the last infusion.

Animal 1, which was edematous before treatment, did not respond to treatment to the same extent as did the other animals. Lead-elimination curves tend to substantiate this observation. Although no kidney function tests were performed, the edema led us to believe that the animal had a nephritis of lead origin.

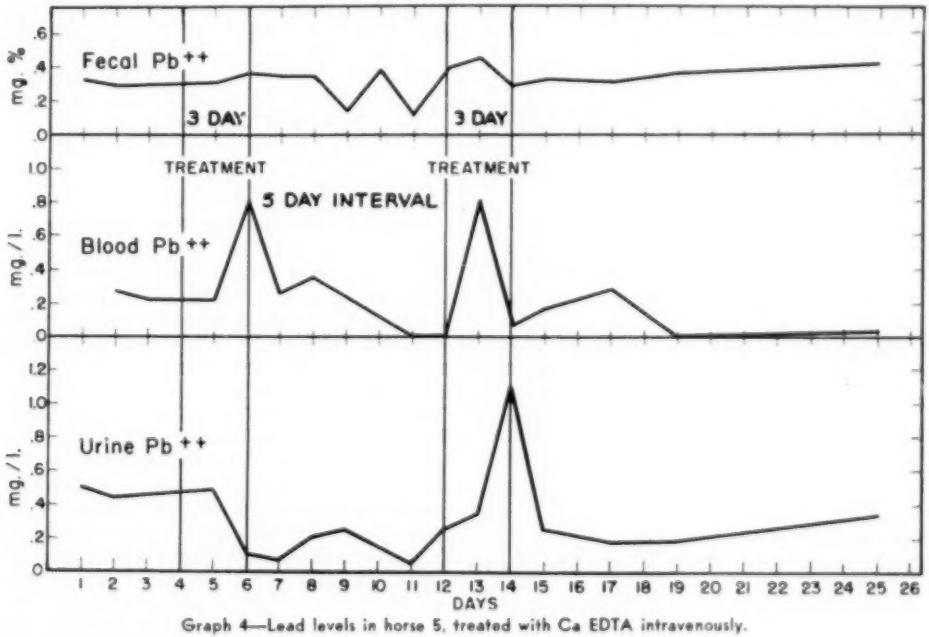
Side effects which have been noted in experimental animals given intravenous Ca EDTA include a fall in blood pressure and temporary respiratory arrest. Dehydration of the subject may occur after several days of injections.[14] Only in animal 1 were side effects observed. This horse first received the injection at a rate of 80 to 100 drops per minute; under this condition, the heart beat became very rapid, respiration became deep and labored, and severe body tremors set in. These signs subsided after the drip rate was decreased to 60 drops per minute. No dehydration was observed in any subject. During one injection, a large amount of the solution was accidentally administered subcutaneously. No tissue reaction was observed.

DISCUSSION

The approach to therapy in lead poisoning in animals has, in the past, been similar to that employed in human patients. Magnesium sulfate has been used to immobilize lead in the gut; calcium salts are used to drive lead to bony depots and may quiet a colicky gut when given intravenously. Sodium citrate in conjunction with a high calcium, high phosphorus diet has been used.[7] BAL (2,3-dimercaptopropanol) has been used as an antilead compound, but reports as to its efficacy and side effects are contradictory.[1,9,10]

Deleading of the bony depots has been accomplished by the use of a low calcium and phosphorus intake, frequently in conjunction with induced acidosis. Potassium iodide has proved effective in deleading, but its mode of action is obscure.

The action of Ca EDTA is largely confined to the dangerous soft tissues. From these tissues, lead is excreted as the lead



Graph 4—Lead levels in horse 5, treated with Ca EDTA intravenously.

disodium salt, which is nontoxic and nonionizable. A certain amount of the lead is driven into the bony depots.

The dosage and concentration of the infused drug used in the above cases were those tested in small experimental animals and were the maximums allowable in these animals. They may not represent therapeutic optimums for horses or other large animals.

When effective dosages are utilized, it has been shown that intermittent dosage schedules produce greater lead excretion than does prolonged constant administration.[14] Daily drips for extended periods produce a refractory state in which lead excretion becomes very low. If a period of time is allowed to elapse after the refractory stage is reached and if therapy is again instituted, lead excretion increases.

No attempt was made to combine the Ca EDTA with acidifying agents which would promote lead mobilization from the bone. From a theoretical viewpoint, the use of the tetra sodium salt alone would be a powerful lead mobilizing agent since it would draw calcium from the soft tissues and promote calcium movement from the bone. This would automatically draw lead from the bone. Such a procedure would require exacting control in order to prevent severe, if not fatal, hypocalcemia.

SUMMARY

Four horses, chronically sick from lead poisoning, were treated successfully with the calcium disodium salt of ethylenediaminetetraäcetic acid. Ca EDTA was diluted with saline to make a 2 per cent solution and was administered intravenously at a level of 1.0 Gm./30 lb. of body weight. At a drop rate of 60 to 80 per minute, no untoward effects were noted.

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Hog Cholera in Ontario

The hog cholera enzoötic, the first in Canada since 1946, affected between 50 and 60 herds and, altogether, about 3,000 hogs were slaughtered and buried in bringing it under control. Since only cooked garbage is fed to hogs in Canada and living virus is barred from the country, the source of the disease is believed to be the trains traveling on an American railroad between New York and Michigan on the Canadian side of Lake Erie. The rare appearances of cholera in Canada are usually in the neighborhood of this railroad. The total average annual cost of hog cholera in Canada is less than the annual cost of vaccinating pigs in some counties in the United States.—*T. Lloyd Jones, D.V.M., Guelph, Ontario.*

Comments on Henny's Essay, "The Hazards of X Rays"

The essay by Henny (October JOURNAL, p. 343) is of real life and death interest to veterinarians. X rays are dangerous, even deadly, but Henny suspects that many veterinarians do not know it. At least they do not act as if they know it. Surely it can not be because they do not care about their own lives.

Veterinarians fluoroscope animals often without warding off the x rays scattered from the animal. They even expose their hands to the direct beam while holding an animal for fluoroscopy; or let an assistant risks his hands that way. Leaded rubber gloves do not protect adequately against the direct beam, although leaded gloves and a leaded apron give fair protection against the scattered x rays. Henny thinks the animal ought to be restrained by mechanical devices and even then no more x ray used than needed.

The total x-ray exposure can be cut down by several strategems, which should be enjoined on fluoroscopists for their own good. Complete dark adaption (15 minutes of red goggles, he says, to which we would add 3 minutes in the dark, by the clock!) can make the eyes 1,000 or 10,000 times as sensitive. High tube voltage increases efficiency of production and of penetration. A filter of 2 mm. of aluminum cuts out a lot of useless soft radiation. A long distance from tube to patient saves the latter's skin from overdosage. A narrow beam saves the fluoroscopist a lot of scattered ray.

In taking radiographs, Henny observes that veterinarians hold the cassette in their hands for the exposure, thus getting the same dose that the animal gets. He thinks this inexcusable. It might be done in an emergency if leaded gloves and leaded apron are worn; however, one good emergency usually leads to another and there is likely to be no end to the accumulation of x-ray injuries possible under such a policy. We like our animal friends but should like our own selves better.

Henny thinks veterinarians do not need to gather their own experience of x-ray injuries and disasters in order to mend their careless ways. He thinks they could make use of what the medical profession has suffered if they would read the record.

We are not too hopeful about this. The trouble is that x-ray injury is not perceptible while it is occurring. Neither is the sunburn while lying on the beach in the springtime. The sunburn breaks out two hours later. The x-ray burn shows up in two weeks. Just as surely as foolish people will get themselves sunburned, so will foolish physicians and veterinarians get themselves x-ray burned. It is time to stop being foolish.— *R. R. Newell, M.D., San Francisco, Calif.*

Certified Meat-Type Swine

To increase meat-type hog production, a plan has been approved by the nation's purebred swine association. Production registry litters to be certified must weigh 200 lb. or more at 6 months of age, and 2 pigs from each litter must be butchered, weighed, and meet certain requirements in length of carcass, depth of back fat, and amount of lean loin. A boar that sires 5 certified litters from different sows is eligible for a registry certificate.—*Farm J., Oct., 1953.*

Tuberculosis in a Cheetah

In February, 1952, a 17-month-old cheetah in the London Zoo became quite ill. Her chief symptoms were a distended abdomen and jaundice. Her cage mate had recently died with generalized tuberculosis and her feces contained acid-fast bacilli. The diagnosis was tuberculosis of the liver. With some difficulty, injections of 0.5 Gm. of streptomycin were given twice daily for twenty days. She was also given 16 Gm. of para-aminosalicylic acid (P.A.S.) on her food for about two months. After a fourteen-day interval, another fourteen-day course of streptomycin was given. Having made an apparent recovery and since her fecal samples were negative, she soon was put back on exhibition.

Eight months later (Dec., 1952) she was again ill with similar symptoms, except for no jaundice and more coughing. Fecal samples were again positive. The animal had now grown until handling was impractical, so streptomycin injections were not attempted. A course of 6 Gm. of P.A.S. plus 150 mg. of iso-nicotyl-hydrazile (I.N.H.) were administered on her food daily. After ten days she quit eating and,

because of dysentery, became very weak. Two weeks later she commenced eating and the oral treatment was resumed. She made an uninterrupted recovery and in March, 1953, her feces being negative, she was again put on exhibition.—*Vet. Rec., July 18, 1953.*

Notes from the Colorado State Association Meeting, Sept. 10-11, 1953

In bovine sterility, the wave of hormone popularity has probably caused the importance of infections in the vagina and cervix to be underestimated. These mild local infections probably produce toxins which kill the sperm.—*G. R. Moore, D.V.M., Michigan.*

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Poor sanitary practices of opposite extremes were found to be reducing breeding efficiency in two dairy herds. In one, the bull was never removed from a pen and was so manure-mired that his sheath was infected. In the other, cleanliness was so emphasized that before each milking all cows were washed with a fire hose, often forcing fecal particles deep into the vagina.—*G. R. Moore, D.V.M., Michigan.*

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Nephritis is much more common in dogs than in other animals. Most cases are infectious and come through the blood stream; very few come from the urinary tract.—*K. W. Smith, D.V.M., Iowa.*

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In acute canine nephritis, antibiotics are often preferable to methenamine. Sulfonamides should be avoided as they may add to the renal embarrassment.—*K. W. Smith, D.V.M., Iowa.*

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In cases of feline urethral obstruction, the calculi are usually within an inch of the end of the penis; some can be relieved by a simple amputation.—*K. W. Smith, D.V.M., Iowa.*

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When a ruminant stops eating, less saliva is secreted to neutralize the acids developed in rumen digestion. The rumen flora, therefore, is affected and under such conditions rumen transplants could not be of much value.—*M. L. Morris, D.V.M., Kansas.*

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Three pounds of glucose placed in a sheep's stomach will cause death in a few hours. An excess of grain will also affect the rumen flora and interfere with digestion.—*M. L. Morris, D.V.M., Kansas.*

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Ponds in ranges and in pastures are often incubators for parasites and diseases.—*A. H. Quin, D.V.M., Missouri.*

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Hospitalized cats often eat best at night. Their diet should be high in fats and proteins, especially gland proteins. It can be low in carbohydrates and minerals. Whole fish is too high in mineral and too low in fat.—*M. L. Morris, D.V.M., Kansas.*

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When 5 of 9 calves with shipping fever died, and none had improved after three days of serum treatment by the owner, the remaining 4 and 8 new cases were treated with streptomycin and sulfamerazine; they responded quickly.—*W. A. Aitken, D.V.M., Chicago.*

Trichinosis in America

The problem of trichinosis in the United States is regarded with astonishment by people in many other countries. According to the director general of the World Health Organization, "When travelers come to the United States from European countries their doctors warn them that under no circumstances should they eat pork of any kind because this country, in that field, is regarded as quite uncivilized. . . . There is no other well-developed country which allows trichinosis among its hogs. . . . In no other country would people be allowed to feed raw garbage to their hogs. . . ."—*Illinois Health Messenger, June 15, 1953.*

Fungous Infections and Antibiotics

Local infections in the mouth with the fungus, *Candida albicans*, have been known for many years but only recently have they become frequent and serious. Three fatal cases following extensive use of antibiotics are reported. In most cases, several antibiotics had been used on the patients but since the increase is coincident with the use of the broad-spectrum antibiotics, they are believed to be most responsible.—*J. Am. M. A., May 16, 1953.*

Corticotropin (ACTH) in Cyanide Poisoning

A healthy man entered by mistake a room filled with hydrocyanic acid gas. When he sank into a deep coma and did not respond to methylene blue, nitrites, and calcium thiosulfate therapy, he was given 25 mg. of corticotropin (ACTH) intravenously. This was repeated in three hours. He did not regain consciousness for twelve hours but showed prompt improvement after each injection of corticotropin.—*J.Am.M.A., Aug. 8, 1953.*

SURGERY & OBSTETRICS
AND PROBLEMS OF BREEDING

Preliminary Observations on the Use of Cortisone in Canine Mast Cell Sarcoma

R. S. BRODEY, D.V.M., V.S.; J. T. McGRATH, V.M.D.; J. E. MARTIN, V.M.D.;
Philadelphia, Pennsylvania

ADRENAL CORTICAL hormones have been reported to exert a favorable, although not a curative, effect on certain neoplasms of man (Taylor *et al.*).[1] In animals, the complete disappearance of multiple mast cell sarcoma in a dog, following treatment with cortisone, has been reported by Bloom.[2] On the basis of Bloom's preliminary report and the known effect of cortisone in reducing the number of mast cells in normal tissues (Asboe-Hansen),[3] cortisone therapy was instituted in 3 cases of canine mast cell sarcoma presented at the University of Pennsylvania, School of Veterinary Medicine.

MATERIALS AND METHODS

Cortisone[3]* (cortone acetate, Merck) was employed in a suspension containing 25 mg. of cortisone per cubic centimeter for intramuscular injection. Tablets containing 25 mg. of cortisone were used for oral therapy.

All cases were biopsied prior to treatment and a histological diagnosis of mast cell sarcoma established. All tissue sections were fixed routinely in a 10 per cent formalin solution and stained with Giemsa, hematoxylin, and eosin.

CASE REPORTS

Case 1.—A 5-year-old male Pointer was presented two weeks after having had several small tumors removed surgically from the left chest wall. On presentation, multiple tumors were observed on the line of incision.

Radical excision of the tumors and axillary node was performed. Histological studies revealed typical mast cell sarcoma (fig. 1). The tumors consisted of densely packed mast cells with occasional bands of a collagenous connective tissue stroma, involving the corium with infiltration into the subcutaneous tissue. There were occasional mitotic figures, some cellular anaplasia, and extreme polymorphonuclear leukocytic infiltration (fig. 2). Metastatic involvement of the axillary lymph gland was present. Within two weeks, multiple tumors recurred at the site of surgery.

On the initial day of treatment, the size of the tumors varied from approximately 2 to 4 cm. in diameter. Treatment consisted of 75 mg. of cortisone daily, intramuscularly. After five days of treatment, the

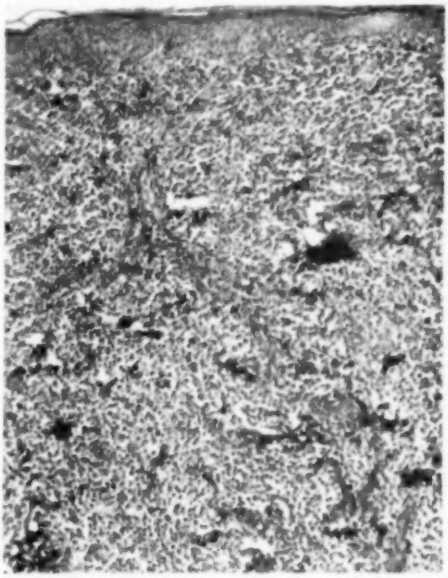

Fig. 1—Typical mast cell sarcoma (case 1). Pretreatment biopsy. 80 x.

Instructor of veterinary surgery (Brodey); assistant professor of veterinary pathology (McGrath); and assistant professor of physiology and pharmacology (Martin), School of Veterinary Medicine, University of Pennsylvania, Philadelphia.

*Cortone acetate Merck, supplied through the courtesy of Dr. L. A. Michaud, veterinary division, Merck and Co., Rahway, N. J.

tumors were noticeably flatter and softer in consistency. Biopsy taken at this time (fig. 3) revealed a marked decrease in the number of neoplastic mast cells and polymorphonuclear leukocytes. There was a relative increase in the amount of collagenous connective tissue, much of which

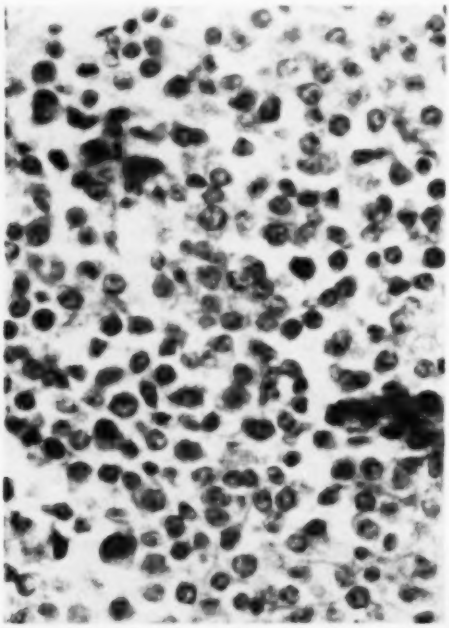

Fig. 2—Mitotic figures, cellular anaplasia, and extreme polymorphonuclear leukocytic infiltration in mast cell tumor (case 1). Pretreatment biopsy. 460 x.

appeared granular and pale-staining in contrast to the sharp, bright homogeneous appearance of normal collagenous tissue. Mast cells observed showed various stages of degeneration; nuclear changes such as hyperchromatism and pyknosis were present concomitant with an indistinct vacuolated cytoplasm and occasional granular clumping (fig. 4). In some sections, there were occasional arterial changes such as thrombosis with hyalinization and periarteritis. In a few areas, the neoplastic mast cells showed no degenerative changes.

Therapy was discontinued for four days and in this period the tumors enlarged markedly and became slightly larger than before treatment was instituted. The dog was then placed on 75 mg. of cortisone intramuscularly twice daily. After nine days, there again was marked reduction in the size of the tumors.

The dog was discharged and maintained on an oral dose of 100 mg. of cortisone daily for two weeks. The animal was then rehospitalized as the tumors had recurred and now involved a large part of the left thoracic wall. Surface ulceration and suppuration were evident and subcutaneous edema was marked. Due to the extensive involvement of the neoplastic process, euthanasia was performed.

Necropsy revealed two large, grayish white, ulcerated tumors of the skin extending from the fourth to the seventh intercostal space and from the eighth to tenth intercostal space. The former measured 12.0 by 10.0 by 3.5 cm. and the latter 7.0 by 9.0 by 3.0 cm. Two smaller, flattened, confluent ulcerated nodules, measuring 3.5 by 3.0 by 1.0 cm., were located on the skin of the right abdominal wall. The adjacent musculature was not involved. Metastasis to the left prescapular node was present. Histological sections of the tumors revealed typical mast cell sarcoma with little evidence of any degenerative processes, as described in the biopsied sections. The adrenal glands were grossly decreased in size. Histologically, there was evidence of cortical atrophy.

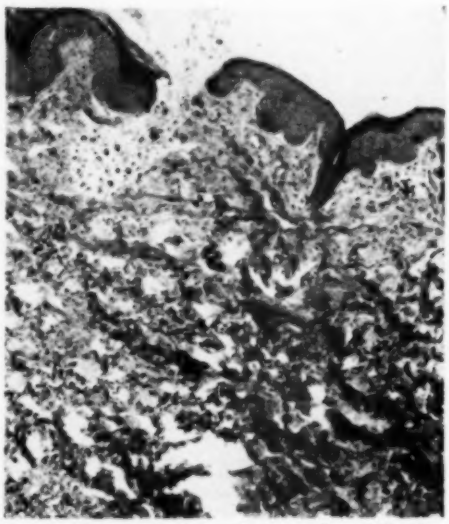

Fig. 3—Biopsy of mast cell tumor (case 1) after five days of treatment with cortisone. Note the marked decrease in neoplastic mast cells and polymorphonuclear leukocytes. 80 x.

Case 2.—A 7-year-old male Chesapeake Bay Retriever was referred by a veterinarian. The right thigh region was diffusely swollen and the overlying skin very tense. On palpation, fluctuation was noted in some areas. There was extreme edema below the hock. In this case, a punch biopsy was performed and a diagnosis made of undifferentiated sarcoma.

During the next ten days, daily intramuscular injections of 100 mg. of cortisone were administered. During this period, the diffuse swelling receded and irregular nodular masses could be palpated in the deeper tissues of the posterior femoral region. There was no evidence of overlying skin involvement.

Biopsy taken at this time revealed a mast cell sarcoma with some degenerative changes as described in case 1.

During the next five days, the dosage was gradually reduced from 100 mg. to 25 mg. of cortisone daily. The animal was discharged and maintained on an oral daily dose of 25 mg. of cortisone for ten days.

The dog was then rehospitalized, as massive tumor growth had occurred. After five days of daily treatment with 175 mg. of cortisone intramuscularly, no reduction in the size of the tumors had occurred and euthanasia was performed.

Autopsy revealed disseminated grayish white masses involving the posterior thigh region, extending from the hock to the tuber ischium. The tumor tissue was ill-defined with litle evidence of overlying skin involvement and was localized in the subcutaneous tissues with extension into the musculature. The femoral vessels were completely encircled by neoplastic tissue. Histological sections revealed typical mast cell sarcoma with no evidence of degenerative changes as noted in biopsied sections. Metastasis was not observed. The adrenal glands were very small and pale, and the cortex atrophied. This was verified on histological examination.

Case 3.—In a 5-year-old female Boxer, with a history of having had a tumor removed from the stifle region, the inguinal lymph node was easily palpable. Multiple tumors appeared on the line of incision three weeks later, and the animal was referred to our clinic for diagnosis. Biopsy revealed a typical mast cell sarcoma.

The animal was discharged and three weeks later rehospitalized as the neoplastic

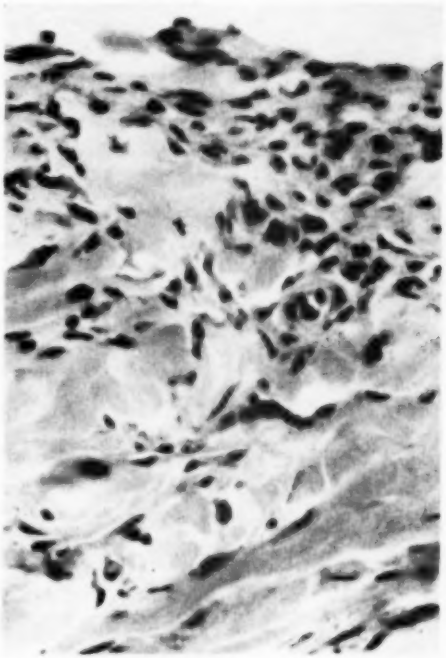

Fig. 4—Mast cells (case 1) showing various stages of degeneration, nuclear changes such as hyperchromatism and pyknosis, concomitant with an indistinct vacuolated cytoplasm and occasional granular clumping. 460 x.

tissue had enlarged to the extent that it almost completely girdled the stifle joint (fig. 5). Surface ulceration, infectian, and edema were present. The main tumor mass measured 9.3 cm. in length and 8.4 cm. in width.

The dog was given daily intramuscular

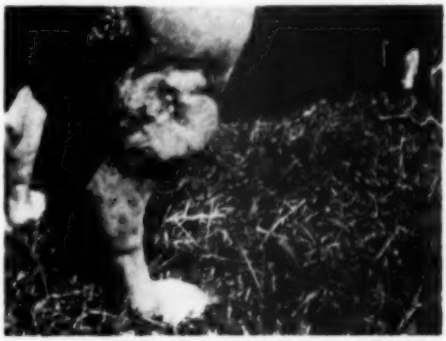

Fig. 5—Dog (case 3) showing tumor on stifle joint.

Fig. 6—Dog (case 3) showing reduction in tumor mass after nineteen days of cortisone therapy.

injections of 100 mg. of cortisone for ten days. Biopsy at this time revealed similar degenerative changes as described in case 1.

The dosage was then reduced to 75 mg. daily for four days and then to 50 mg. for five more days. At this time, the tumor mass measured 6.0 cm. in length and 4.5 cm. in width, and the leg had almost returned to normal size and appearance except for some surface ulceration (fig. 6). The dog was discharged and kept on an oral maintenance dose of 25 mg. daily for two weeks.

The animal was then rehospitalized as the tumor had reached massive proportions (fig. 7). Euthanasia was performed.

Fig. 7—Two weeks after figure 6 was taken, the tumor (case 3) had reached massive proportions.

Necropsy revealed a large cauliflower-like growth with an ulcerative, crater-like center, involving the skin of the lateral posterior region from the right stifle joint extending to a level just above the hock. The mass measured 18.0 by 15.0 by 5.4 cm. On dissection, tumorous tissue extended from the hock to the vulva and involved, predominantly, the skin and subcutaneous tissues of the medial aspect of the thigh. Metastasis was noted in the right inguinal region, external iliacs, pelvic and right renal lymph nodes as well as the liver, spleen, and kidneys. Histological sections revealed typical mast cell sarcoma.

The adrenal glands showed some decrease in size with focal areas of atrophy evident in the zona fasiculata of the cortex.

SUMMARY

The use of cortisone in the treatment of 3 cases of canine mast cell sarcoma is described.

In all cases, there was an initial dramatic reduction in the size of the tumor; however, in each case, decreasing the dosage or cessation of therapy was followed by marked increase in tumor size and eventual euthanasia.

Biopsies taken during cortisone therapy revealed a marked decrease in the number of neoplastic mast cells, with various degenerative changes present.

The use of cortisone resulted in various degrees of adrenal cortical atrophy.

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In preparing a Brahman bull for surgery, it may be advisable to withhold water for twenty-four to forty-eight hours so that he will drink water containing a sedative, either chloral hydrate or nembutal.—*A. M. Mills, D.V.M., Athens, Ga.*

A Case of Osteomyelitis of Facial Bones in a Dog

D. V. BENSON, D.V.M., M.S.; A. A. CASE, D.V.M., M.S.; LOREN D. KINTNER, D.V.M., M.S.

Columbia, Missouri

A 6-year-old male Schnauzer was brought to the University of Missouri veterinary clinic on Oct. 30, 1952.

The case history revealed a slowly developing serous nasal discharge occasionally showing a tendency to be purulent. This had been developing for three months. In the three weeks previous to its presentation to the clinic, two veterinarians had treated the dog. The owner reported that one gave a single injection of penicillin and the other dispensed antihistamine tablets. Neither treatment brought any relief.

On our advice, the dog was left at the clinic for examination and laboratory tests. The blood count showed: 4,670,000 r.b.c., 14,750 w.b.c., 2 per cent eosinophils, 9 per cent monocytes, 23 per cent lymphocytes, and 66 per cent neutrophils (20% juveniles, 46% segmenters). Wright's stain of the nasal discharge revealed many neutrophils and no eosinophils. *Staphylococcus albus* and *Brucella bronchoseptica* were cultured from the nasal exudate. Radiographs showed atrophy of the turbinate bones, especially on the left side, and degeneration of the anterior end of the nasal septum (fig. 1). The patient had a mildly depressed attitude but the temperature was normal.

The treatments given did not affect the course of the disease. These included penicillin and streptomycin, intramuscularly; aureomycin, intravenously; terramycin, intravenously; and 0.25 per cent of neosynephrine and bacitracin nose drops. Aureomycin nose drops were used for one day but were not well tolerated so were discontinued. During treatment the temperature remained normal, the patient gradually became more depressed, and the nasal discharge gradually increased in amount and became more viscous in nature but not more purulent.

On December 6, it was noted that the skin over the center of the frontal bones, near their union with the parietal bones, fluctuated with breathing. This is the central area outlined by white tape in figure 2. Palpation revealed a small area with complete absence of bone beneath the skin.

The owner consented to an operation

Fig. 1—Radiograph showing atrophy of the turbinate bones, especially on the left side, and degeneration of the anterior end of the nasal septum.

Fig. 2—Area (marked by white tape) where the skin over the frontal bones fluctuated with the dog's breathing.

From the School of Veterinary Medicine, University of Missouri, Columbia.

which was to terminate with euthanasia if surgery revealed a continued unfavorable prognosis. On December 12, an operation was performed which revealed almost complete destruction or resorption of the left turbinate bones, a large portion of the

Fig. 3—The frontal bones have been removed to show the resorption of the left turbinate bones, a large portion of the right turbinate bones, and the anterior one-third of the nasal septum.

right turbinate bones, and the anterior one-third of the nasal septum. There was open communication between the left and right nasal cavities. Euthanasia was considered advisable. In figure 3, the surgical opening in the frontal bones has been enlarged and most of the nasal bones removed for photography. Sections of the skin over the affected and adjoining area showed only inflammatory changes. There was no indication of a neoplasm. Sections of the bones remaining in the area showed osteomyelitis.

It was not possible to determine the etiology and pathogenesis which resulted in the above described condition. Among the possibilities to be considered are primary sinusitis, infected teeth, a blow to the head resulting in fractures, and a foreign body in the nasal cavity.

Transportation and Bull Fertility

The fertility of 36 bulls, of six breeds, was compared before and after being transported by railroad or truck for 300 to 1,000 miles. Their average fertility in the first service, prior to moving, was 66.8 per cent, the first month after moving, 69.2 per cent, and the second month 68.4 per cent. Apparently, no detrimental effect can be attributed to transportation.—*J. Dai. Sci. June, 1953.*

Gangrene Following Intraärterial Transfusion.—Intraärterial transfusion resulted in gangrene in 1 human patient. The perforated artery had been ligated after the transfusion was completed. It is suggested that hemorrhage can be controlled without ligating the artery if a gauze sponge is applied to the vessel at the site of puncture, with firm pressure for five minutes after the needle is withdrawn.

The wound can then be closed with mattress sutures in the skin and further firm pressure applied.—*J. Am. M.A., June 27, 1953.*

Influence of Thyroprotein on Lactation

Twenty, first-calf Holstein-Friesian or Jersey heifers were fed thyroprotein (1 or 1½ Gm./cwt./day) from the fiftieth postpartum day until ninety days before their next expectant parturition. This method of feeding continued during each lactation as long as the cows were in the herd; 4 completed five lactations and 3 completed six. Eight similar heifers served as controls.

Thyroprotein caused an immediate increase in milk production and butterfat in practically all animals, but the increase was only transient and did not result in a greater milk production over an entire lactation. The body weight decreased more in the cows fed thyroprotein but a rapid increase occurred immediately after withdrawal of the thyroprotein. When allowance was made for body weight changes, these cows were less efficient than normal cows during their first lactation. Their efficiency con-

tinued to decrease in subsequent lactations while that of the control cows remained constant. The mortality of calves born to cows fed thyroprotein was higher. Apparently, feeding thyroprotein to dairy cows is not a desirable practice.—*J. Dai. Sci., July, 1953.*

Surgical Correction of Fecalith in a Pony

CLIFTON N. MURPHY, D.V.M.

Stillwater, Oklahoma

On Feb. 9, 1953, a 1-year-old, 225-lb., Shetland filly was referred to the veterinary clinic of the Oklahoma A. & M. College by a practicing veterinarian.

Tenesmus and anorexia had been present for four days. Two days previously the pony had been treated with ½ gallon of mineral oil orally and trocharization. Since there was no response, a tentative diagnosis of intestinal obstruction was made.

The symptoms were: temperature 101 F., the mucous membranes congested, the sclera injected, pulse rapid and thready, marked dehydration and depression, gait stiff and stilted, abdominal muscles tense and sensitive, respiration fast and shallow. The rectum was empty.

Surgery, in place of more drastic purgatives, was decided upon. The pony was restrained on the right side and a light surgical anesthesia produced with 30 cc. of equitol[R] intravenously. The left paralumbar fossa was prepared for surgery and desensitized with a 2 per cent procaine solution.

The skin was incised, then each muscle layer was split in the direction of its fibers and the peritoneal cavity was invaded. Upon abdominal exploration, the middle section of the small colon was found to be obstructed and grossly distended. The involved portion, which was exposed through the incision by gentle traction, was approximately 3 inches in diameter. The anterior portion contained flatus and some fecal material while the area posterior to the obstruction was collapsed and empty.

No intestinal incision was made since the material causing the obstruction could be flaked by external pressure. The exposed portion of the colon was kept moist with warm saline packs, then replaced and a routine laparotomy completed. Crystalline sodium penicillin (1,000,000 units) and dihydrostreptomycin (0.5 Gm.) were given intramuscularly daily for five days.

Approximately three hours after surgery, the pony showed signs of abdominal pain and became restless. A warm soapy enema was given and oily feces were passed. One hour later, it passed feces and more of the mineral oil that had been administered two days previously.

The morning following surgery, the animal's appetite and bowel movements were normal. The pony's temperature remained normal and it made an uneventful recovery.

From the School of Veterinary Medicine, Oklahoma A. & M. College, Stillwater.

Enterotomy in a Thoroughbred Foal

ERIC F. PALLISTER, V.S., D.V.M., M.S.

Philadelphia, Pennsylvania

Retention of meconium occurs more often in foals from stall-fed mares. When a foal is 12 hours old, if there has not been an elimination from the bowel, it is advisable to make a rectal examination with the finger. The presence of a firm mass in the posterior bowel would indicate the necessity for remedial measures; these usually consist of digital manipulation, the administration of milk of magnesia, cascara sagrada, or castor oil *per os* and gentle enemas. It should always be borne in mind that the rectum of the newborn foal is a particularly delicate structure and tolerates only the most gentle manipulation. The obstruction is caused by hardened masses of meconium in the colon and rectum. Colicky pain in a newborn foal should always suggest digital examination *per rectum*.

On April 14, 1953, a call was received at the clinic of the School of Veterinary Medicine, University of Pennsylvania, from an equine practitioner requesting assistance in handling a case of persistent retained meconium in a 2-day-old Thoroughbred foal. It was reported that oil *per os*, and repeated high enemas, had proved fruitless; the foal was becoming depressed and uneasy, and an emergency laparotomy was deemed imperative.

Dr. Pallister is assistant professor of veterinary surgery, School of Veterinary Medicine, University of Pennsylvania, Philadelphia.

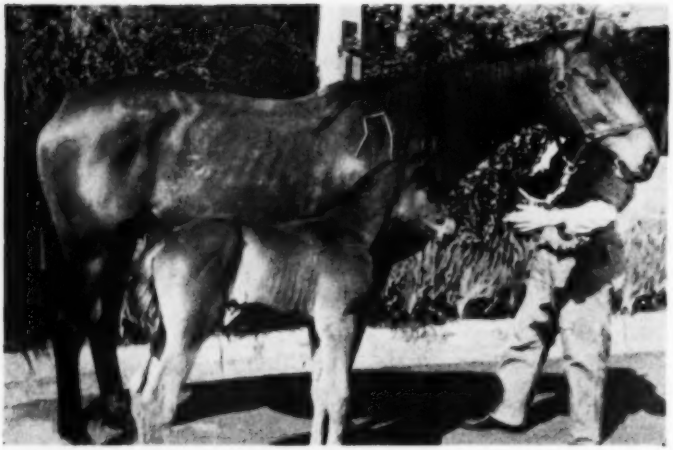

Fig. 1—Foal (and its dam) five days after enterotomy

The foal with its dam was transported by truck to the large animal clinic. Physical examination indicated that the foal was apparently a full term, normal individual. It manifested a slightly elevated tail, some depression, uneasiness, and colicky pain. He would nuzzle at the mare's nipples half-heartedly, but would not nurse; then he would lie in dorsal recumbency.

Digital examination *per rectum* revealed a firm mass anterior to the brim of the pelvis. Enemas were expelled as clear in color as when introduced into the posterior bowel, and tenesmus had failed to deliver any excreta. Attempts were made to break down the mass and relieve the obstruction with forceps. This too proved ineffective and slight damage to the mucosa was indicated by the presence of blood on the forceps.

Laparotomy and possible enterotomy was the elected procedure. Anesthesia was obtained with sodium pentobarbital by the intravenous route, to effect. Physiological saline with glucose 5 per cent solution was administered by drip intravenously throughout the operation. Restraint was in dorsal recumbency. The site was prepared in conventional manner for aseptic approach by paramedian incision lateral to the prepuce. A longitudinal incision was made through the skin, the musculature was divided by separation of muscle fibers, and the parietal peritoneum incised. Exploratory palpation revealed a firm, well-formed mass approximately 12 inches in length, occupying the terminal portion of the bowel. Anterior to this, there were several firm spherical masses which distended the colon greatly, but there was no gas present in the bowel.

Enterotomy was achieved by a 6-inch longitudinal incision in the small colon, opposite the mesenteric attachment, approximately 18 inches anterior to the pelvic portion of the bowel. The mass was removed intact by direct traction with forceps, supplemented by a milking action with the gloved hand. The spherical masses were extricated in a similar manner through the same incision.

The bowel was closed with one row of Lembert suture using No. 1 medium chromic catgut, and this was reinforced with a row of Connell suture. The peritoneum and tranversus muscle were approximated with simple continuous suture and the remainder of the abdominal wall with a sliding mattress-type suture. The skin apposition was achieved with No. 1 dermalon suture, using interrupted vertical mattress sutures and placing a small gauze drain at one end of the incision. Aureomycin powder was dusted lightly into the wound as closure progressed.

Crystalline penicillin, 1,000,000 units, and dihydrostreptomycin, 0.5 Gm., were administered intramuscularly.

Within twenty-four hours, there was some fecal excretion and the foal had an uneventful convalescence. The cutaneous sutures were removed and the case discharged on the eighth day.

Incidence of Diseases and Mortality Among Two Breeds of Egyptian Sheep

H. REDA, M.V.Sc., and M. M. OLOUFA, Ph.D.
Giza, Egypt

Of all the Egyptian livestock, sheep have the most distinct breeds. The Ossimi and Rahmani are the most popular. Both breeds are of the fat-tailed type with the rams having strong horns, while the ewes are hornless. The Ossimi has a narrow body with white, coarse fleece of medium length, and a reddish brown head. The Rahmani is brownish red, with coarse wool which becomes grey as the animal grows older.

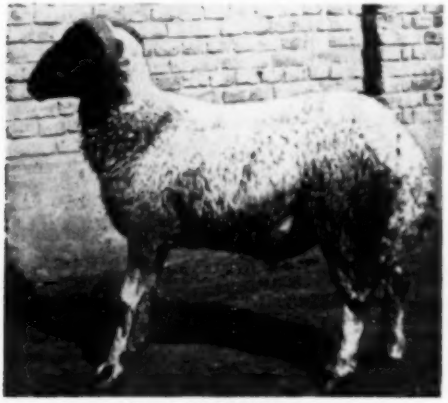

Fig. 1—An Ossimi ram.

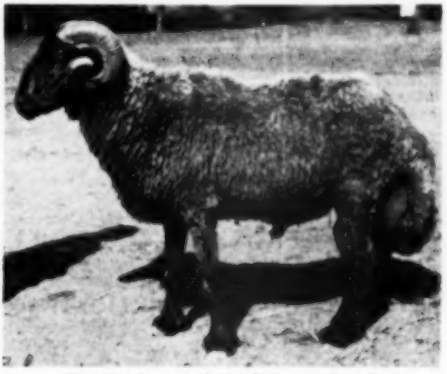

Fig. 2—A ram of the Rahmani breed.

In sheep, as in other animals, instances of differential resistance to diseases and parasites are found. In California, Stewart *et al.*[3] have described experiments that indicate breed variations in susceptibility to the parasite *Ostertagia circumcincta*: The Romneys were highly resistant, the Rambouillets less so, and the Downs were most susceptible. In Egypt, Reda[2] found the percentage of enzootic pneumonia higher in the Rahmani than in the Ossimi sheep. Nichols[1] stated that the Romney March are recognized to have a relatively high immunity to foot rot, just as the Shetland breed of sheep is resistant to ticks.

The present study was to investigate the comparative susceptibility to diseases of Ossimi and Rahmani sheep. A total of 755 Ossimi and 727 Rahmani sheep were included in this study (1948 through 1952).

Results and Discussion.—The incidence of disease varied from year to year but averaged 29.6 per cent in the Ossimi and 39.1 per cent in the Rahmani. The mortality averaged 11.5 per cent in the Ossimi and 17.7 per cent in the Rahmani. The differences between the two breeds in both the incidence of disease and mortality were highly significant.

TABLE I—Incidence of Disease and Mortality Among the Ossimi and Rahmani Sheep During 1948 Through 1952

	Ossimi					Rahmani				
	Total	Diseased		Mortality		Total	Diseased		Mortality	
Year	(No.)	(No.)	(%)	(No.)	(%)	(No.)	(No.)	(%)	(No.)	(%)
1948	125	37	29.5	11	8.7	133	49	36.9	19	14.2
1949	145	48	33.1	15	10.4	155	58	37.4	21	13.5
1950	143	37	26.8	10	6.9	137	52	37.9	16	11.6
1951	154	45	29.2	11	7.1	153	60	39.2	17	11.1
1952	188	57	30.3	40	21.2	149	59	39.5	56	37.5
Total	755	224	29.6	87	11.5	727	278	39.1	129	17.7

In 1952, there was a relatively high mortality in both breeds due to an experiment in which many ewes were bred to

Drs. Reda and Oloufa are lecturers, animal breeding department, College of Agriculture, Giza, Egypt.

lamb during the summer, an unfavorable season for parturition under local conditions. Pneumonia, enteritis, and ascites were the main factors contributing to the mortality of lambs.

TABLE 2—Causes of Diseases and Mortality in Ossimi and Rahmani Sheep

Diseases	No. animals diseased		No. animals dead	
	Ossimi	Rahmani	Ossimi	Rahmani
Pneumonia	25	35	20	35
Enteritis	28	34	21	34
Ascites and debility	26	32	16	32
Impaction	11	12	8	9
Metritis	5	7	2	4
Jaundice and liver flukes	27	30	16	9
Indigestion	20	21	....	1
Diarrhea	9	18	....	....
Uremia	....	....	3	3
Liver diseases	....	....	1	2
Rheumatism	9	9	....	....
Udder diseases	7	16	....	....
Eye diseases	6	6	....	....
Wounds	20	23	....	....
Lameness	13	8	....	....
Fractures	4	12	....	....
Mange	14	15	....	....
Total	224	278	87	129

These studies show the Ossimi sheep to be less susceptible to disease than the Rahmani in Cairo and places of similar climate.

References

[1]Nichols, J. E.: Livestock Improvement. Oliver and Boyd, Edinburgh (1947): 43.

[2]Reda, H.: Contagious Pneumonia in Sheep and Allied Cases. Thesis for M.V.Sc. degree, Faculty of Veterinary Medicine, Cairo, 1945.

[3]Stewart, M. A., Miller, R. F., and Douglas, J. R.: Resistance of Sheep of Different Breeds to Infection by *Ostertagia Circumcincta*. J. Agric. Res., 55, (1937):923.

Penicillin Transmitted by Placenta

Thirty parturient women were given intramuscular injections of 400,000 units of penicillin when they entered the delivery room. During delivery, serum from an arm vein of the mother and from the umbilical cord were compared for penicillin content. Three hours after the injection, the penicillin content of the serum from the infant was 60 per cent of that of the maternal serum. It is assumed that the penicillin passed through the placenta according to the laws of diffusion. Soon after the injection, penicillin appeared in the amniotic fluid, having been excreted through the kidneys of the infant, the concentration being higher than in serum of the infant.—*J. Am. M. A., July 4, 1953.*

Intramedullary Fixation of Femoral Fracture in a Chinchilla

D. A. PRICE, D.V.M.

Sonora, Texas

Recently, a fine male chinchilla jumped from a man's hands to the ground, with resulting fracture of the left femur. Palpation revealed what appeared to be a short oblique fracture in the distal third of the bone, with a considerable amount of over-riding. An unusual situation was pointed up by the following facts: We do not conduct a practice and, therefore, had access to none of the common fixation materials; we did not know whether the patient, being a rodent, would tolerate, for long, either an unfixed fracture or external fixation; the nearest practitioner likely to possess intramedullary equipment of suitable size for a 15-oz. animal was not only 100 miles distant but his city was inaccessible as a result of a tornado that very day.

Fig. 1—This photograph was taken ten days after intramedullary fixation of the left femur.

Obviously, extemporaneous measures were indicated if a valuable animal was to be salvaged, so fixation was attempted by the following means within three hours of the accident. Satisfactory anesthesia was obtained by the intraperitoneal injection of 4 ml. of 7 per cent chloral hydrate solution per kilogram of body weight (dosage based

From the Ranch Experiment Station, Sonora, Texas.

on some previous experimental work with guinea pigs). The fur was clipped from the entire limb with scissors. Following the application of a skin disinfectant, a small incision was made over the trochanteric fossa. A ¼-inch electric drill fitted with a 1/64-inch bit was used to drill a hole through the trochanteric fossa into the medullary canal. The bit was then withdrawn and a straight, 5-inch, cutting-edge surgical needle was used as an intramedullary pin. The needle was guided into the distal fragment by palpation alone and was firmly driven into the compact substance at the distal end of the bone. The needle was then cut off at the level of the trochanteric summit. The skin incision was so small that closure by collodion was sufficient.

The patient placed some weight on the affected limb the following day. Crystalline penicillin was administered during the first four days, after which time he could move about the cage in an almost normal manner. At the end of two weeks, a callus had formed, but a red serous discharge was noted at the site of the needle insertion. When pus appeared after eighteen days, the needle was removed and the subsequent recovery was uneventful.

Our gratifying experience with this fracture is related because it illustrates that although the size of these valuable furbearers may at times necessitate improvisations, the results can be rewarding.

Optimum Time for Insemination.—A number of crossbred cows in estrus were inseminated three times, twelve hours apart, using semen from Holstein-Friesian, Guernsey, and Aberdeen-Angus bulls so that the sire of the calf could be identified. Of the 15 calves resulting, 6 were from the first insemination, 7 from the second, and 2 from the third.—*J. Dai. Sci., June, 1953.*

Site of Semen Deposition Compared.—First service conceptions were attained in 53 per cent of 103 heifers when the semen was deposited at the os uteri, in 68 per cent at the mid-cervix, uteri, and in 65 per cent when deposited in the uterus.—*J. Dai. Sci., June, 1953.*

Award for Research on Infertility

The American Society for the Study of Sterility announces its 1954 contest for the outstanding contribution to the subject of infertility and sterility. The award is $1,000 and the essay will appear on the society's program in 1954. Essays must be received by March 1, 1954. For further particulars, write Dr. Herbert H. Thomas, secretary, 920 South 19th St., Birmingham, Ala.

Parthenogenesis in Turkey Eggs

For the first time in birds, embryos have been discovered developing in unfertilized eggs. When the eggs of Beltsville Small White turkey hens which had been isolated three to six months earlier, and two months before they were mature enough to come into production, were incubated, 15 per cent developed embryonic tissue. The same thing occurred when a later group was isolated at 4 to 12 weeks of age, without ever contacting mature males.

The parthenogenic development starts slowly and continues for only a few days. It is not visible to the candler until about the fifth day, whereas a normally fertilized egg would reach that stage in eighteen to twenty-four hours of incubation. A few embryos develop almost normally but in 98 per cent the development was unorganized and simply a translucent membrane. It may be related to a hormone imbalance. Most turkey hens do not produce such eggs but others produce many. This discovery might have significance in cancer research. —*Agric. Res., August, 1953.*

Contrast Medium Dosage

An error in contrast medium dosage appears in table 1 (JOURNAL, Sept., 1953:190) in the article "A Method of Visualizing the Urinary Tract and a Basis for Assessing Renal Function in Small Animal Radiography" by E. J. Bishop. In the last column, the "cc./lb. of body weight" applies only to the first dosage. All succeeding amounts are the total dosage of contrast medium.

CLINICAL DATA

Endocrine Diseases of the Dog Associated with Hair Loss

Sertoli Cell Tumor of Testis, Hypothyroidism, Canine Cushing's Syndrome

DAVID L. COFFIN, V.M.D., and TODD O. MUNSON, V.M.D.

Boston, Massachusetts

IT HAS LONG been recognized that hair is one of the most sensitive reflectors of health in both animals and man. The hair not only is affected in local processes of the skin, such as bacterial infection, mange, contact dermatitis, and allergic states, but is also changed in nutritional deficiencies and systemic disease.

One of the most readily recognized skin conditions is the loss of hair, or alopecia. A special type of alopecia, in which the hair loss is bilaterally symmetrical, is seen in dogs. This condition is not especially common, but an understanding of its pathogenesis and etiology will go far toward simplifying differential diagnosis of skin diseases in the dog. Although several types of this state exist, they have certain common features. The hair loss is the same on both sides of the body. It occurs at the sites of wear, from contact of the dog with various objects, where he scratches himself frequently, and at the area of contact with the harness or collar if he wears one. The characteristic dog has bare sides, flanks, anterior surfaces of the limbs, a bare area behind each ear, and a bare tail with a tassel of hair on the end. The hair gradually becomes sparse on the back and, if the dog survives long enough, he will become completely hairless. The hair can be pulled out readily, even at the sites where it appears fairly abundant. There is no pain or pruritus associated with this type of hairlessness, unless it is complicated by an infectious or an allergic condition.

The bilateral distribution of these lesions results from several facts: (1) The hair follicles become atrophic and inactive, resulting in the cessation of the normal cycle of shedding and regrowth. (2) The hairs remain in the follicles until they are removed by some mechanical abrasive factor. (3) Because the dog is a bilaterally symmetrical animal, the abrasion is distributed evenly, at any given site, on opposite sides of the body.

However, due to the selectivity of the dog's actions, the lesion is not diffusely applied to the whole side but is concentrated at certain specific sites which, in general, appear to be the same in most individuals. Therefore, the sequence of the alopecia follows a definite progression with the development of a distinctive pattern which is modified only by an idiosyncrasy of behavior on the part of an individual animal or because of some act of his master, such as grooming or requiring the animal to wear a special harness. The actual diffuse nature of the follicular lesions is admirably illustrated when such an animal is subjected to clipping before any hair changes have become apparent. In these instances, the animal's coat, which had previously been luxuriant, permanently remains as stubble after the clipping.

As the disease progresses, the areas first showing hairlessness become larger, new areas become bare, and eventually the dog may become almost entirely bald. The hair of the face, head, and feet usually appears more persistent.

Three endocrine conditions, namely (1) Sertoli cell tumor of the testis, (2) thyroid deficiency, and (3) a pituitary-adrenal state, here termed "canine Cushing's syndrome," are the subjects of this discussion. While the hairlessness associated with testicular tumors and hypothyroid states has long been known, the syndrome referable to the pituitary-adrenal disease, so far as we know, has not been hitherto reported

*Dr. Coffin is pathologist, Angell Memorial Animal Hospital; instructor in pathology, Harvard Medical School; research associate in pathology, Children's Hospital, Boston. Dr. Munson is a staff member, Angell Memorial Animal Hospital, Boston.

in the literature available to us. The state of the hair and skin, differing slightly in these conditions, will be discussed in detail under each syndrome.

SERTOLI CELL TUMOR

The Sertoli or sustentacular cell of the testis appears to be the male counterpart of the granulosa cell of the ovary. Tumors derived from this cell, in male dogs, are known to produce estrogenic hormone,[1] an excess of which produces hairlessness and feminization.[2] Review of the pertinent literature and more detailed discussion of the pathology of this condition have been published elsewhere.[3,4] The condition is seen in dogs of middle age or beyond (4 to 16 years, average 10 years[3]) in which there are bilateral hair changes as previously mentioned, thin, smooth skin, and sometimes slight pigmentation. Hypertrophy of the nipples and mammary glands and relaxation of the penile sheath and perineal fascia are also evident (fig. 1). Many of these dogs are sexually attractive to other males.

The tumors are usually unilateral and frequently arise in a cryptorchid testis (29 % occurred in cryptorchids[3]). The testis containing the tumor may be unrecognizable, existing merely as a tumor mass, while the opposite testis is extremely atrophic. Since only tumors large enough to be palpable appear to produce endocrine symptoms, diagnosis can usually be made by palpation.

The tumorous specimen presents a whitish, firm, slightly greasy cut surface which

Fig. 1—Wooly mongrel with Sertoli cell tumor syndrome. There is characteristic bilaterally symmetrical hairlessness on the neck, sides, flanks, buttocks, and tail. Note the enlarged nipples and the presence of the protuberant tumor mass at the external inguinal ring.

Fig. 2—French Poodle with bilaterally symmetrical hairlessness due to hypothyroidism. Note the similarity of the pattern to figure 1. Hair grew on this animal following administration of thyroid extract.

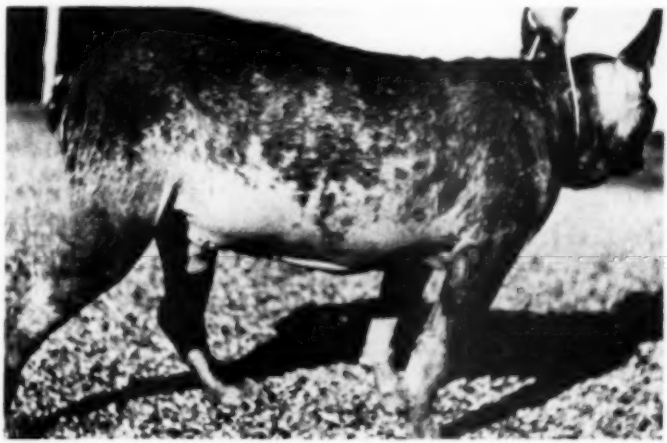

Fig. 3—Boston Terrier with the canine Cushing's syndrome. The hair has disappeared from behind the ear, the belly, the sides, and areas of the legs. The remaining hair is broken and bristley. Note the pendulant abdomen and relaxed, skeletal-braced position of the front quarters.

Fig. 4—Boston Terrier showing the hyperkeratosis sometimes seen in the canine Cushing's syndrome. The buttocks and posterior surfaces of the thighs are shown.

does not bulge. The prostate may be either increased or decreased in size. If enlarged, it frequently contains cysts which may communicate with the urethra, causing discolored or odoriferous urine.

HYPOTHYROIDISM

The fact that hypothyroidism alters the character of the hair in both animals and man is so well known that no special documentation is required here. In dogs, hair loss due to hypothyroidism is seen in individuals of all ages and in males, females, and castrated animals of both sexes.

The onset is gradual; the hair loses its luster and begins to wear away in the typical bilateral sequence (fig. 2). It is recognized most frequently in fat animals but may occur in nonobese, and even thin, animals. The behavior is sluggish and there is loss of libido in noncastrates. The skin is smooth, thin, and tends to be pigmented on the bare areas. The remaining hair is fine or fuzzy.

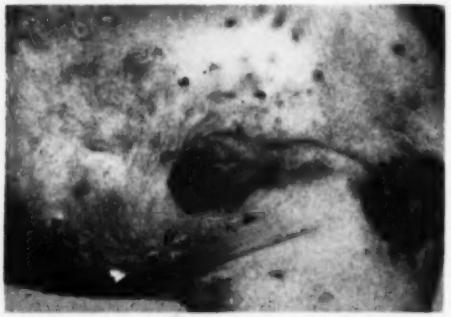

Fig. 5—Boxer with the Cushing's syndrome, showing the vesicle formation and focal pigmentation following their regression.

CANINE CUSHING'S SYNDROME

This condition occurs in dogs of middle age or beyond (6 to 16 years). It predominates in Boston Terriers (table 1). The onset is insidious, being marked by gradual enlargement of the belly and roughness of the coat. As the disease progresses, the belly continues to enlarge and, as the flesh is lost elsewhere, tends to have a pendulous appearance. Concomitantly, the hair-loss sequence progresses in a typical, bilateral pattern. The legs develop bare areas sooner than in the two previously mentioned conditions (fig. 3). The remaining hair is rough, dry, often broken, and may easily be pulled from the follicles. Muscular weakness appears, being marked by the dog's trembling and by the assumption of a straight-legged, skeletal-braced posture. Muscular relaxation contributes to the pot-bellied appearance. The skin is often rough and scaly because of the presence of minute blackened projections (fig. 4). Small pustules or blisters frequently occur sporadically, especially on the belly. When these evacuate, the site is subsequently marked by ragged rims of desquamating epidermis. Characteristically, the centers of these areas become blackened, due to an accumulation of melanin pigment

TABLE 1—Breed, Age, and Sex of 15 Dogs with Cushing's Syndrome

Breed	No.	Age	M.	F.
Boston Terrier	9	6-12	6	2*
Terrier mongrel	1	12	1	
Wire-Haired Terrier	1	13		1
German Shepherd	1	14	1	
Spitz	1	11	1	
Dachshund	1	3		1
Boxer	1	9	1	

*One spayed female.

(fig. 5). The surface of the skin in such dogs is often cool.

In the majority of cases, polydipsia and polyuria occur to a degree compatible with diabetes insipidus.* The specific gravity of the urine may range as low as 1.002 to 1.005 (table 2). There is lymphopenia and eosinopenia to a sufficient degree to be diagnostic when other symptoms are suggestive (table 3).

TABLE 2—Blood and Urine Chemistry in 9 Dogs with Cushing's Disease. Blood Sugar and Urea Nitrogen Are in Milligrams per 100 cc. of Whole Blood; Potassium (K) and Sodium (NA) in Milliequivalents per Liter of Serum

Urine			Blood			
Specific gravity	Albumin	Sugar	Blood sugar	Urea nitrogen	K	NA
1.012	neg.	neg.	77	—	—	—
1.002	neg.	neg.	76	—	—	—
1.030	neg.	neg.	97	23	5.6	154
1.005	neg.	neg.	81	73	5.9	—
—	—	—	—	44	5.5	—
1.005	neg.	neg.	90	14	4.4	158
1.004	neg.	neg.	—	—	4.1	135
1.014	±	—	—	—	—	—
1.005	+	neg.	—	—	—	—

TABLE 3—Blood Picture of 12 Dogs with Cushing's Syndrome. Hemoglobin Is in Grams per 100 cc. of Blood; Leukocytes (w.b.c.) in Number per Cubic Millimeter; Other Elements in Percentage

Hb.	w.b.c.	Neut.	Eos.	Bas.	Lymph.	Mon.
10.3	2,900	89	0	0	5.0	6.0
13.4	27,000	95	0	0	2.0	3.0
16.4	14,600	85	0.25	0	6.7	6.0
13.0	18,300	91	0.5	0	3.5	2.5
12.5	15,300	89	1.0	0	4.0	6.0
13.0	14,300	87	0	0	2.0	10.0
14.8	16,700	82	0	0	5.0	10.0
18.0	9,200	81	1.0	0	2.0	14.0
16.4	10,400	85	1.0	0	4.0	10.0
—	—	88	0	0	1.0	8.0
—	—	86	0	0	5.0	9.0
—	9,700	86	2.0	0	6.0	6.0

Hb = hemoglobin; w.b.c. = white blood cells; neut. = neutrophils; eos. = eosinophils; bas. = basophils; lymph. = lymphocytes; mon. = monocytes.

The pathology and endocrine mechanism and its relationship to Cushing's disease in man will be discussed in detail elsewhere.[6] The disease in dogs is caused by a disturbance of the pituitary-adrenal mechanism, in which there are basophil tumors of the pituitary gland (fig. 6) and secondary symmetrical cortical hyperplasia of the adrenal glands (fig. 7). We believe that increased production of adrenal cortex hormone produces the diagnostic reduction of the lymphocytes and eosinophils, as well as changes in the skin and hair which will be discussed below. No other abnormal biochemical findings referable to the endocrine disease have thus far been noted among our cases (table 2).

The pathology consists, essentially, of the following: (1) enlargement of the pituitary with adenomatous growth of the basophil cells, and frequently multiple cysts, leading to pressure on, and secondary atrophy of, the posterior lobe (probably explaining the diabetes insipidus on the basis of diminution of the antidiuretic hormone produced there*); (2) symmetrical hyperplasia of both adrenal cortices with the adrenals four to eight times the normal weight (fig. 7); (3) atrophy of the hair follicles, sebaceous glands, and epidermis; and (4) loss of dermal fat, condensation of the collagenous and elastic fibers, and hyperkeratosis.

GENESIS OF ENDOCRINE ALOPECIA

Hair changes associated with endocrine dystrophies have been the subject of much speculation. Hairlessness is often ascribed to "endocrine dysbalance" without attempting to determine the specific endocrine organ involved. While the subject is complex, it probably is an understandable "endocrine dysbalance."

It is hoped that by reviewing the known causes, estrogenic tumors of the testis and hypothyroid states; also by reporting the existence of a third cause, the pituitary-adrenal syndrome on the basis of our clinical and pathological material, that other syndromes which exist may also be distinguished and classified for a better understanding of the total situation.

The possible biological mechanisms responsible for the skin and hair changes may be portrayed diagramatically (fig. 8). Estrogen from the Sertoli cell tumor acts either (A) directly on the skin[7] or, possibly, (B) in conjunction with adrenal-cortex hormone in a synergistic effect,[8] causing atrophy of the hair follicles and sebaceous glands, and thinning of the epithelium. A second less

*Diabetes insipidus was present in a case of adiposo-genital syndrome in a dog due to an infundibuloma of the hypothalamus and pituitary, reported by Saunders, Stephenson, and McEntee.[5] They cite five additional reports of pituitary or hypothalmic tumors in which diabetes insipidus was present. Two were accompanied by the adiposo-genital syndrome. They cite 3 additional cases of adiposo-genital syndrome in dogs. The characteristics of this condition, namely generalized obesity and genital atrophy, are unlike those of the canine Cushing's syndrome described here.

*See preceeding footnote.

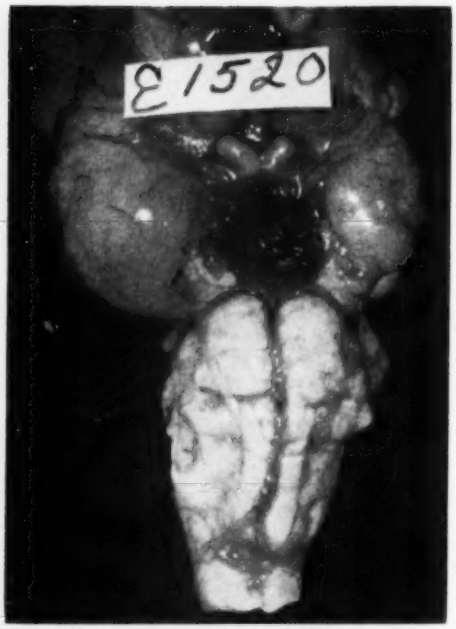

Fig. 6—Boston Terrier with the Cushing's syndrome. The greatly enlarged pituitary body appears on the ventral surface of the brain below the optic chiasm as a prominent dark mass.

likely possibility exists as follows: estrogen acts either by (C) stimulation of the anterior pituitary gland which in turn stimulates the adrenal cortex, or by (D) direct stimulation of the adrenal cortex. The skin changes, according to this hypothesis, would be the result of the effect of augmented corticosterone. Evidence for the direct action of estrogen and against the alternate possibilities of action through stimulation of the adrenal gland are: (1) known experimental production of skin change by direct action of estrone,[3] and (2) that in our material no significant secondary hyperplasia of the adrenal cortices was present and no lymphopenia or eosinopenia characteristic of adrenocortical hyperplasia occurred in the few cases of Sertoli cell tumors for which we have blood pictures.

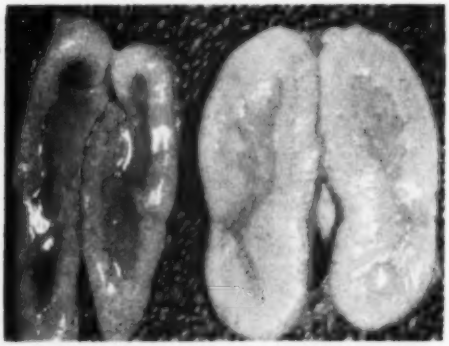

Fig. 7—Boston Terrier with the Cushing's syndrome showing the symmetrical enlargement of the adrenal cortex (right) as compared with an adrenal from a normal Boston Terrier of the same age and approximately the same weight.

The mode by which the hair changes are produced in the canine Cushing's syndrome may be explained as follows: augmented adrenocorticotrophic hormone from the anterior pituitary, probably resulting from cytological change there (hyperplasia or tumor), stimulates the adrenal cortex to hyperplasia and increased output of corticosterone which acts directly on the skin by alteration of nutrition and other changes, causing atrophy of the epidermis.

Similar phenomena have been experimentally produced in rats by the parenteral administration of adrenocortotropin[9] and by percutaneous administration of adrenocortical preparations.[10] Experimental work suggests that the adrenocortical hormone of the C-11 oxygenated group is most active in the production of the above effects.[11] It would appear that the C-11 oxygenated hormones are augmented in our dogs, since a strong hypercortical effect exists, as indicated by the degree of lymphopenia and eosinopenia (table 3). The absence of the abnormal blood potassium and sodium levels (table 2) indicate that the C-11 nonoxygenated steroids are not significantly augmented.

As some of our males suffered slightly from hypogonadism, no increase in androgenic steroid was expected. No hyperestrogenic changes have been detected. In the rat, at least, only the C-11 oxygenated group appears under the regulation of the pituitary gland and this hormone is known to be produced in the zona fasciculata of the adrenal gland. It is this zone that is most hyperplastic in our dogs. The exact means by which adrenocortical hormone acts on the skin has not yet been elucidated. It is known that the administration of corticosterone of the C-11 oxygenated group in high dosage causes breakdown in protein and loss of nitrogen by excretion.[12] Ablation of the adrenals is accompanied by a rapid increase in hair growth over normal, accompanied by an appreciable increase in oxygen consumption of the skin. Thus, it would appear that the nutrition of the skin is in some way diminished by adrenal cortex hormone.

THERAPY

Therapy has been successful in stimulating hair growth and counteracting the other changes in the Sertoli cell tumor syndrome and the hypothyroid state. No treatment used thus far, however, has in

any way altered the course of the canine Cushing's syndrome.

Sertoli Cell Tumor.—It has been the routine practice at the Angell Memorial Animal Hospital to remove the tumorous with the nontumorous testis. Following castration, there is cessation of most of the symptoms referable to the hyperestrogenic effect of the Sertoli cell tumor. The attractiveness to other males ceases within a few days. There is a gradual improvement in tone of the penile sheath and perineal tissues. Hair growth is usually apparent within four to six weeks on nude areas or where the hair has been clipped but no diminution in the size of hypertrophied nipples appears to occur. In one case where metastasis of a Sertoli cell tumor occurred, temporary improvement, after castration, was followed by return of all the symptoms as the metastatic tumors grew in the lumbar lymph nodes.[4]

Hypothyroidism.—The administration of thyroid extract in doses varying from ½ to 3 gr. daily has been successful in overcoming the hairlessness due to hypothyroidism. The precise schedule must be worked out by trial with the end point being an increased state of vigor in the animal without undue trembling or nervousness. Signs of hair growth may be expected in four to six weeks, as in the Sertoli tumor syndrome. There is also a change in the character of the hair; coarser hair replacing the fuzzy type characteristic of the hypothyroid state.

Cushing's Syndrome.—Several hormone preparations have been employed empirically in the Cushing's state. These include androgen, estrogen, thyroid extract, and desoxycorticosterone acetate. No improvement has been apparent. There is some indication that irritating ointments or other skin trauma promote slight hair growth temporarily, but they have absolutely no effect upon the other component of the syndrome. The only treatment that

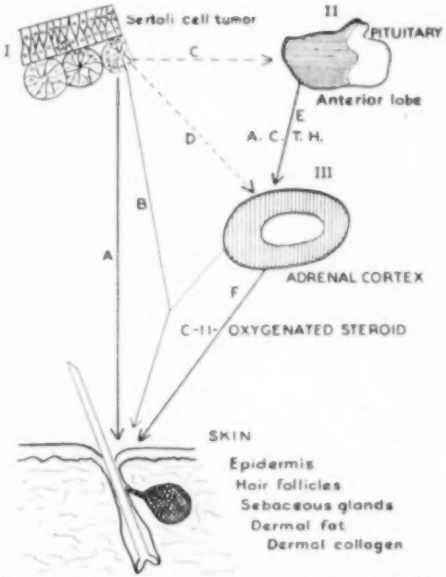

Fig. 8—Schematic representation of the probable mechanisms by which hairlessness is produced in the Sertoli cell tumor syndrome and the Cushing's syndrome.

Sertoli cell tumor I secretes estrogen which reacts directly on the skin and hair follicles (A). Other possible modes are synergistic action with adrenocorticosterone (B), or indirectly by stimulating the anterior pituitary (C) or the adrenal cortex (D).

Basophilic tumor of anterior pituitary II secretes ACTH (E) which stimulates adrenal cortex III to augment the excretion of corticosterone (probably C-11-oxygenated steroid) to react on the skin and associated structures (F).

TABLE 4—Differential Diagnosis in Dogs with Endocrine Alopecia

Sertoli cell tumor	Cushing's syndrome	Hypothyroidism
Bilateral, symmetrical hairlessness; hair pulls out; does not shed.	Similar to Sertoli-cell tumor.	Similar to Cushing's syndrome; onset more gradual.
Skin smooth, thin.	Skin crusty, blackish papilla or scales, blisters with pigmented centers; surface cold.	Skin smooth and thin.
Tumors in scrotal or cryptorchid testes. Opposite testis and penis atrophic; gynecomastia.	Testes normal or slightly atrophic.	Slightly hypogonad.
Normal muscular activity.	Weakness, trembles, pot-bellied, not obese.	Usually a little sluggish.
Attraction for males; sometimes aggressive.	Eosinopenia, lymphopenia, usually diabetes insipidus.	May be normal, thinner than normal or obese.

would appear to be rational would be some method to reduce the output of the pituitary gland, such as radiation therapy or surgical removal of a portion of the adrenal tissue.

We have not employed x-ray irradiation of the pituitary gland but feel that it should be tried. One dog was subjected to unilateral adrenalectomy but, unfortunately, did not survive the operation. In many cases, the most troublesome symptom in the Cushing's state is the presence of diabetes insipidus, causing discomfort to the animal and annoyance to the owner. Control of this symptom has been achieved by the injection of small doses of the posterior pituitary hormone, pitresin. The pitresin in oil is used and the client is instructed to inject the material at home. For Boston Terriers a dose of ¼ cc. is usually sufficient to reduce noticeably the fluid intake and amount of urination for two to four days. The client is advised to vary the frequency of the dose to achieve the desired effect.

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Streptomycin and the Eighth Nerve

To study the effect of streptomycin and dihydrostreptomycin on pulmonary tuberculosis, two similar groups of 34 patients each were treated daily for 120 days. One group was given 1 Gm. of streptomycin sulfate, the other group 1 Gm. of dihydrostreptomycin sulfate daily. Six of the first group had vestibular disturbances manifested by a transient minimum vertigo but none had auditory disturbances. In the latter group, 2 had vestibular disturbances and 5 had auditory disturbances.—*J.Am.M.A., Aug. 22, 1953.*

[The *J. Am. M.A.* (Aug. 15,1953: 1574) reports that in a much heavier dosage of streptomycin, the loss of hearing was often 50 per cent complete. It did not progress after six months following the discontinuance of the drug, but the condition was permanent. No dizziness or vertigo was noticed.]

Cortisone for Arthritis in a Bull

A Shorthorn bull developed progressive arthritis, especially in the hock and hip joints. Treatment over a period of time with sodium salicylate and also by diathermy was ineffective. He could scarcely walk, exhibited pain, and was losing weight. Cortisone acetate was provided for experimental treatment. Two grams were injected intramuscularly followed by 1 Gm. daily for ten days. Improvement was noticed by the fifth day, and, by the tenth, he was walking and eating normally and could be used for service. However, by the eleventh day after the treatment ended, he had returned to the original condition; therefore the cortisone injections were repeated. Sodium salicylate was also given in his food and in six days the bull was almost normal but, again, stiffness returned fourteen days after the last treatment. He was destroyed but no autopsy was possible. —*Vet. Rec., Aug. 8, 1953.*

A Possible Bacteriophage Active Against *Moraxella (Hemophilus) Bovis*

A Preliminary Report

W. L. KANAWYER, D.V.M.; A. EISENSTARK, Ph.D.; E. J. SPLITTER, D.V.M., M.S.

Manhattan, Kansas

THE INCONSISTENCY of experimental transmission of bovine infectious keratitis with *Moraxella bovis* has deterred complete understanding of this disease of the bovine eye. Various investigators[1,2] have reported that this disease can be reproduced by *M. bovis*. However, even though identical material is inoculated into both eyes of an animal, frequently only one eye becomes infected. When such inconsistent and variable results were encountered in our studies of the transmission of bovine infectious keratitis, some type of interference was suspected. Specifically, the following question was raised: Why should one eye of a calf be sensitive and susceptible to *M. bovis* while the other eye remains resistant?

In the course of these studies, a filtrate of eye washings from calves produced typical phagelike plaques on plate cultures which had been seeded with *M. bovis*. Since the presence of bacteriophage offered one possible explanation of the peculiar experimental transmission described above, further investigation was undertaken. A preliminary report of the procedure and results of this phagelike action is reported.

PROCEDURE

Lacrimal secretions were collected from 2 calves which had previously been inoculated by briskly rubbing corneal and conjunctival epithelium with *M. bovis*. The disease failed to develop in the eyes of either calf. Secretions from the inoculated eyes were Seitz filtered and one drop of filtrate was placed on the surface of a two-layer Difco tryptose agar plate prepared by the method of Delbrück[3] and seeded with *M. bovis*. The strain of *M. bovis* employed had previously appeared to reproduce cases of bovine infectious keratitis. Plates were incubated at 37 C. for twenty-four hours.

RESULTS

A circular, clear area occurred at the point on the plate where the drop of filtrate had been placed. This procedure was repeated on several plates with similar results.

These cleared areas were scooped out, suspended in broth, filtered, and the filtrate titrated. The plaque count of this filtrate was 5×10^9 per milliliter, indicating that multiplication of the original agent had occurred.

Further studies are in progress to characterize the bacteriophage and to determine its source. The possibility that the phage arrived in the eyes through lysogenic strains of *M. bovis* will be investigated also.

SUMMARY

Examination of filtrates from eye secretions of 2 calves indicated that bacteriophage might be present, as evidenced by the production of clear zones on the surface of *Moraxella bovis*-seeded plates. This evidence was further substantiated by titration of the material in the clear zones. The titer of this material was 5×10^9 bacteriophage particles per milliliter, using the plaque count method.

The presence of such bacteriophage may explain some of the inconsistent and variable results in transmission of bovine infectious keratitis.

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Contribution No. 134, Department of Veterinary Medicine, and No. 284 Department of Bacteriology, Agricultural Experiment Station, Kansas State College, Manhattan.

Treating Swine Erysipelas.—In field trials on the treatment of acute swine erysipelas, penicillin gave a recovery rate of 95 per cent within twenty-four hours, and was much superior to swine erysipelas antiserum which produced a recovery rate of 80 per cent within three days.—*Vet. Bull., May, 1953.*

Observations of an Outbreak of Vibriosis in Sheep

G. BISWAL, D.V.M., M.S.; H. E. RHOADES, M.S.;
P. B. BARTO, V.M.D., M.S.; C. C. MORRILL, D.V.M., Ph.D.

Urbana, Illinois

THIS OUTBREAK of vibriosis occurred in a flock of 114 yearling Hampshire ewes on a farm in DeWitt County, Illinois. They were purchased in September, 1951, from a shipper who had bought them in Montana. Ninety Hampshire ewes from the same shipment were sold to a neighbor who reported no abortions in his flock. Western ewes had been purchased in 1949 and 1950 through the same shipper with no history of abortions.

Suffolk rams purchased in Illinois during the three previous years were placed with the ewes in September. Abortions began occurring the second week of January, 1952. A total of 32 (28%) of the ewes aborted. In addition, some lambs dropped at term were weak and some of these died within a week after birth. Thirteen ewes failed to lamb. Sixty-nine ewes had normal lambs; of these, 5 had twins. The ewes showed no apparent clinical symptoms other than abortion. Retention of the placenta was not observed in any of the aborting ewes. A blood-tinged mucous discharge from the vulva continued for a few days after each abortion.

Eighteen bred Karakul ewes were allowed to run with the Hampshires. There were no abortions among the Karakuls. These had been purchased in Illinois, Colorado, Oklahoma, Wisconsin, and New Mexico.

There were no abortions during the spring of 1953. The live lamb crop was 137 per cent with a loss of 10 per cent of the lambs after birth attributed to chilling, pneumonia, or starvation.

MATERIALS AND METHODS

Twelve aborted fetuses were submitted to the diagnostic laboratory, College of Veterinary Medicine, University of Illinois, for examination and diagnosis. *Vibrio fetus* was isolated from these fetuses.

In order to study the lesions of vibriosis, 2 ewes, from the same flock, in an advanced stage of pregnancy were selected for panhysterectomy. Both ewes reacted to the *V. fetus* agglutination test. The fetuses, uteri, and membranes were examined. One normal healthy ewe in the same stage of pregnancy from an uninfected flock was also examined in the same manner. The following material was streaked onto tryptose-crystal violet plates: contents of the stomach, small intestine, cecum, colon, and from the kidney, lungs, heart blood, brain stem, amniotic fluid, allantoic fluid, fetal membranes, and the umbilical cord of the fetus; also the intercaruncular area of the uterus and placentome of the dam. Inoculated plates were incubated at 37 C. under 10 per cent carbon dioxide. Tissues from the same organs collected for histopathological study were fixed in Zenker's fluid (Ferrari modification). The processes of dehydration, clearing, and paraffinization of the tissues were completed on an autotechnicon. Bone marrow was fixed in 10 per cent neutral formol-saline for Levaditi staining. Tissues were sectioned at 4 to 6 μ, stained with hematoxylin and eosin, and mounted in clarite.

Semen was collected from 3 rams of the infected flock for bacteriological examination. The method of electrical stimulation of ejaculation of rams as originally developed by Gunn[4] was utilized.

Two pregnant guinea pigs and 2 pregnant rats were injected intraperitoneally with the stomach contents of aborted lamb fetuses.

A single batch of concentrated antigen was prepared for conducting the tube-agglutination tests. This antigen was composed of equal parts of the strain of *V. fetus* isolated from this outbreak and a bovine strain. Tryptose agar slants were inoculated separately with the two cultures and incubated for seventy-two hours at 37 C. under 10 per cent carbon dioxide. The growth was washed off and suspended in physiological saline containing 0.5 per cent formalin. Antigen was diluted as needed with the formolized saline to a turbidity equivalent to tube No. 1 of the McFarland nephelometer.

Serum dilutions were prepared with the diluted antigen and incubated at 37 C. for forty-eight hours. The tests were conducted on each animal in the flock at monthly intervals for ten months. Serums from 10 uninfected ewes from another flock were used as negative controls.

RESULTS

Gross Pathology.—Edematous areas were

From the Department of Veterinary Pathology and Hygiene, University of Illinois, Urbana.

The authors express their appreciation to Dean R. Graham for his coöperation in pursuing this work; to Dr. H. J. Hardenbrook for performing the hysterectomies; to Dr. U. S. Garrigus for his interest and coöperation in this problem; and to Mr. Orville Willemssen and Mr. R. T. Hamilton for their technical assistance.

observed in the fetal membranes. The placentomes were dull and soft as compared to the normal which were firm and glistening in appearance. Upon incision, the placentome characteristically showed a dark center with dark streaks extending toward the periphery. The caruncular diameter was 18 to 21 mm. as against 12 to 16 mm. in the normal. The mucosa was covered with dark brown fluid containing partially clotted blood. The infected uterine wall was thickened; the cut surface revealed marked edema.

In most of the cases, the fetal stomach was filled with reddish brown, slimy fluid containing flakes. The stomach contents of an occasional fetus were clear. The peritoneal, pleural, and pericardial cavities contained blood-tinged fluid. Subcutaneous edema was not observed.

Microscopic Pathology.—The uterine glands were mildly infiltrated with leukocytes. In some of the cells, the cytoplasm was vacuolated as a result of which the nuclei were pushed toward the lumen. Invading leukocytes, predominantly polymorphonuclears, were occasionally discernible within these vacuoles. Mild periglandular cellular infiltration was evident. A moderate endometrial edema was present. Eosinophils were scattered throughout the wall but were possibly a little more numerous in the muscular layer.

The pathological changes within the placentomes were striking. The maternal septums appeared thickened. The giant cells (diplokaryocytes) lining the walls of the maternal septums were large and numerous. Most of these giant cells were multinucleated. The vessels in the maternal septums were engorged. Besides a few normal extravasations, greater accumulations of blood were found within the spaces between the fetal villi and the maternal crypts. Within these extravasations it was not unusual to find phagocytic giant cells (fig. 1). The fetal villi and the trophoblastic cells lining the walls of the maternal septums of the uterine part of the placentome were necrotic as evidenced by pyknosis. The degree and distribution of this pathological change were variable from one placentome to another and even within different parts of the same placentome. Edema in the interior of the placentome was prominent. Frequently, hematogenous pigments were found in these locations.

The fetal hepatic parenchymal cells showed a varying degree of degeneration, characterized by a mild to moderate degree of vacuolation suggestive of a fatty change, and an occasional focus of coagulation necrosis (fig. 2). Hemorrhage within the cortical labyrinth was observed in the kidney (fig. 3). The pulmonary bronchioles were filled with fine granular materials

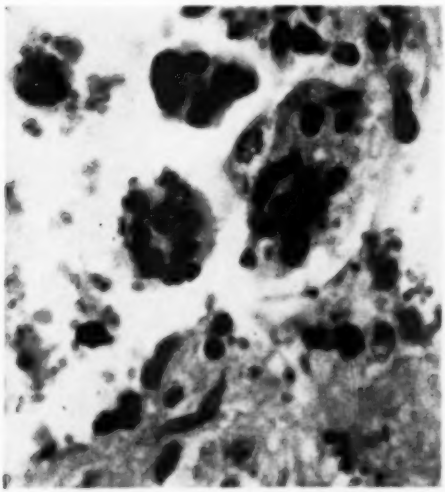

Fig. 1—Phagocytic giant cells. Hematoxylin-eosin stain. 210 x.

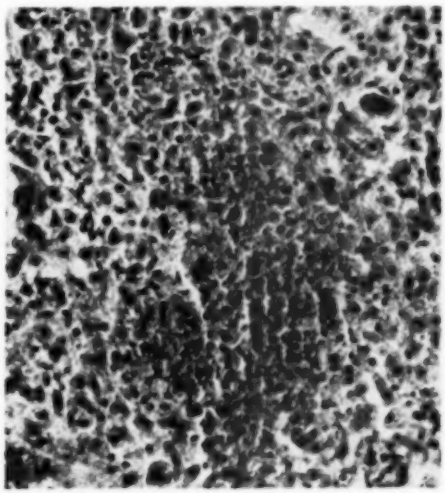

Fig. 2—Focal coagulation, necrosis of the liver. Hematoxylin-eosin stain. 210 x.

and degenerated epithelial cells; the alveoli contained plasma protein, as evidence of pulmonary edema, and a few mononuclear phagocytes. Acute congestion of the vessels with mild, rather diffuse, leukocytic infiltration was observed in the fetal membranes. A mild to moderate degree of edema was present in the chorion.

No pathological changes were observed in the rest of the organs. *Vibrio fetus* was not observed in any of the tissue sections.

Pathogenicity.—When 2 pregnant guinea pigs and 2 pregnant rats were injected intraperitoneally with the fresh stomach contents of aborted ovine fetuses known to contain *V. fetus*, none aborted and *V. fetus* could not be reisolated.

Serology.—The highest titer recorded for a known *V. fetus*-infected ewe was 1:400. Control ewes showed no reaction at a dilution higher than 1:25. The titers remained relatively constant for ten months after the outbreak. Some of the animals in the outbreak developed a titer before abortion, others afterward. *Vibrio fetus* was recovered from the fetus of 1 ewe having a negative titer. Two ewes reacting at a high titer lambed normally. All sheep reacted negatively to the Brucella plate-agglutination test.

DISCUSSION

MacFadyean and Stockman[7] reported that in infected flocks of sheep the abortion rate would average 23.2 per cent. The abortion rate in the flock under observation was 28.0 per cent. MacFadyean and Stockman[7] and Smith[11,12] were of the opinion that retention of the placenta was a characteristic symptom of the infection. No case of retained placenta was observed by the authors. Ryff[10] stated that a few cases of abortion took place in the early part of pregnancy. Most of the aborted fetuses examined by the authors were near full term. The time of introduction of the infection may have influenced this. The present outbreak occurred in an enzoötic form corroborating the observations made by Welch and Marsh.[14]

Plastridge and Williams[8] reported a high incidence of vibrionic abortion in cattle, the diagnosis being based on the agglutination test. Blakemore and Gledhill[1] stated that the titer was highest immediately after abortion and a multiplicity of antigenic types is possible. Levi[6] observed that the titers obtained varied according to the strain used in the preparation of the antigen. He found that serums from infected ewes reacted to a higher titer against an antigen prepared from the infecting strain than those isolated from other outbreaks.

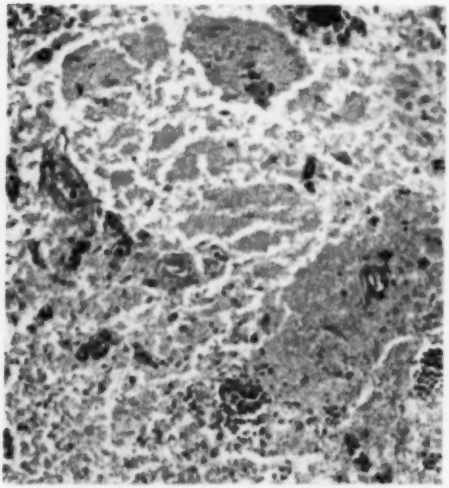

Fig. 3—Hemorrhage in the renal cortical labyrinth. Hematoxylin-eosin stain. 100 x.

Fig. 4—Giant cells associated with purulent endometritis. 430 x.

In this outbreak, a significant agglutination titer did not always appear in the ewes known to have aborted from *V. fetus* infection. Also, positive reaction to the agglutination test in ewes lambing normally indicated that infection did not always result in abortions. Failure of the correlation of positive titers with abortions in every case raised the question whether the time of infection may have accounted for this variation. According to Plastridge *et al.*,[9] 90 per cent of the cows that reacted positively to the blood test became negative within six months. The titers in this flock of sheep remained relatively constant over a ten-month postabortive period.

MacFadyean and Stockman[7] isolated the organism from the uterine exudate, membrane, and the fetus. Plastridge and Williams[8] reported the isolation of the organism in cattle from the stomach contents of the fetus, uterine fluid, amniotic fluid, and cotyledons. In our studies, organisms were isolated from the contents of the stomach, small intestine, cecum, colon; and from the kidney, lungs, heart blood, brain stem, amniotic fluid, allantoic fluid, fetal membranes, and the umbilical cord of the fetus; also the intercaruncular area of the uterus and placentome of the dam.

Stegenga and Terpstra[13] reported the isolation of *V. fetus* from the semen of a bull suspected of spreading infection. Easterbrooks and Plastridge[2] concluded that vibriosis could be spread through artificial insemination with *V. fetus*-infected, diluted semen. Our limited attempts to isolate the organism from the semen of rams reacting to the agglutination test failed.

Gminder[3] produced abortion in pregnant guinea pigs by subcutaneous injection of one or two loopfuls of culture. Lerche[5] induced abortion in pregnant guinea pigs by injecting cultures intraperitoneally and by placing cultures in the conjunctival sac. In our experiment, pregnant guinea pigs and rats injected intraperitoneally with the stomach contents of aborted fetuses known to contain *V. fetus* did not abort, and *V. fetus* could not be isolated from the animals when killed.

However, when a suspension of *V. fetus* of bovine origin was injected subcutaneously into 6 guinea pigs, abortion did occur in one instance and *V. fetus* was isolated from the uterine discharge but not from the liver and spleen of the guinea pig. The uterus contained about 0.5 ml. of a mucoid, cloudy fluid. The uterine wall was thickened and the mucosa hyperemic, with a few petechiae and several necrotic patches about 0.5 cm. in diameter. An agglutination test run with the blood collected from the aborting guinea pig reacted at a dilution of 1:80. The same strain of *V. fetus* used in the inoculation was used as the antigen.

Liver, kidney, spleen, and sections of the uterus from the aborting guinea pig were saved for histopathological examination. Parenchymal cells of the liver were markedly distended, possibly due to glycogen content. However, the presence of some rather clear-cut vacuoles suggested the presence of some fat also. The distention appeared slightly less marked at the periphery of some lobules but was diffuse. The kidney showed mild or early focal interstitial nephritis characterized by small foci of lymphoid infiltration and one rather large area of fibrosis. There were cystic dilatations of the uriniferous tubules in the fibrotic area and in the papilla distal to it. The fibrotic area might have constituted an old infarct. The spleen appeared somewhat hyperplastic. No other significant lesions were noted. Much of the uterine mucosa was intact and essentially normal. Other areas, however, showed infiltration with a few neutrophils. An occasional small area showed a frankly purulent endometritis (fig. 4). Near the areas showing other evidence of inflammation, there was a tendency toward giant cell formation (fig. 4). Marked swelling and probably some proliferation of vascular endothelium were observed. One large vein contained a clot which might well have been antemortem, although this point was not certain.

The pathological process observed in the ovine placentomes was characterized by the thickening of the septums, increase in the number of nuclei in the diplokaryocytes lining the septums, increased extravasation of blood, and edema. Occurrence of a few eosoinophils in the uterine muscular layer of the infected ewes is not explained. Pulmonary edema, occasional hepatic focal coagulation necrosis, and renal hemorrhage were observed in the fetal lambs dead of vibrionic infection.

SUMMARY

1) An enzoötic outbreak of ovine vibrio-

sis is described. The abortions, which reached an incidence of 28 per cent, occurred in late pregnancy. Not a single case of retention of placenta was observed in any of the aborting ewes.

2) *Vibrio fetus* was isolated from the contents of the stomach, small intestine, cecum, colon; and from the kidney, lungs, heart blood, brain stem, amniotic fluid, allantoic fluid, fetal membranes, and the umbilical cord of the fetus; also the intercaruncular area of the uterus and placentome of the dam.

3) The pathological changes observed in naturally infected pregnant ewes and experimentally infected pregnant guinea pigs were chiefly confined to the uterus and placenta.

4) The agglutination titers remained relatively constant for ten months after the outbreak. The highest titer recorded for a known *V. fetus*-infected ewe was 1:400.

5) There were no abortions during the year following the outbreak.

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Reactions to Antibiotic Troches.—A controlled study of several hundred patients with minor oral disorders indicated that a severe glossitis or stomatitis occurred after taking antibiotic troches five times per day for four days. Mild reactions occurred in 14 per cent of 100 control patients who were given placebos, whereas mild to severe reactions occurred in 51 per cent of those given chlortetracycline (aureomycin) troches; in 38 per cent given troches with penicillin; and in 53 per cent given troches with terramycin. The reactions were limited to the mouth and pharynx.—*J.Am.M.A., July 25, 1953.*

Chlortetracycline for Actinomycosis

Seven cases of actinomycosis were treated with 750 mg. of chlortetracycline (aureomycin) orally every six hours for ten days, then 500 mg. every six hours for eighteen days. Six cases with cervicofacial actinomycosis, 3 of which followed extraction of teeth, responded readily. The seventh patient was critically ill with a hepatic abscess. In a few months, this patient also was working and had regained his normal weight. There were no recurrences.—*J. Am. M.A., Sept. 5, 1953.*

Pullorum disease today is often more acute in adult chickens than in young ones. —*H. Van Roekel, D.V.M., Amherst, Mass.*

Teat Vesicles as Primary and Almost Exclusive Lesions in an Extensive Outbreak of Vesicular Stomatitis (New Jersey Strain) in Milking Cows

PIETRO STROZZI, D.V.M., and TEODORO RAMOS-SACO, V.M.D.

Lima, Peru

IN MAY, 1952, dairy cattle of Casma and Huarmey valleys were affected by an outbreak of a vesicular type of disease with lesions localized, in milking cows, almost exclusively in the teats, mouth lesions being exceptional.

The disease spread rather rapidly, covering an extensive area of the country in approximately fifteen days, passing through a great number of dairy farms, and finally reaching the Lima valley which is the largest dairy section of this country. Although the morbidity was high with regard to the area covered, rarely were entire herds affected as is the case with foot-and-mouth disease.

The time elapsing from the moment that the infection was initiated in a herd until the last case of infection has varied, sometimes being as long as a month and a half. Milking cows were affected almost exclusively, it being observed only exceptionally in dry cows, bulls, heifers, and calves. The disease spread rapidly, decreasing gradually after reaching its highest peak.

While lesions were localized in the teats in milking cows, in the other animals they were observed in the mouth cavity.

Lack of certain facts makes it impossible to explain the way in which the virus spreads from one farm to another. On the other hand, spread of the virus within a herd seems to be favored by factors that facilitate the transportation of the virus from the teat lesions of 1 animal to another.

There are some indications that milking practices play an important role in spreading the disease. For instance: (1) The disease is much more frequent in milking cows than in dry cows, bulls, and calves; (2) the primary location of the lesions is in the teats; and (3) in a herd where some cows were milked by hand and others by machine, the disease started in the group milked by hand and spread rapidly within this group, while the other group was affected at a much later date.

It is possible, also, that a type of small biting fly (simuliid), observed in great numbers on several infected farms, may play a part in spreading the virus.

SYMPTOMS AND CLINICAL COURSE OF THE DISEASE

We believe we are confronting an atypical form of vesicular stomatitis. Usually the first signs of the disease go unnoticed, but when a more thorough examination is made, the great majority of cases show slight dullness, decreased appetite, slight fever, and decreased milk production twenty-four or forty-eight hours before the external signs of the disease are evident.

The most striking symptom is the presence of primary vesicles on one or more teats. The vesicles develop in different number and size but only on the surface of the teats. We have never observed them on other regions of the udder. When the vesicles start to show, usually the entire teat becomes tumefied and the skin surrounding the lesions is inflamed and painful. Most of the vesicles become lacerated the first day during milking. Those that are protected from mechanical injury remain intact five or six days until the walls become thin enough to burst spontaneously, leaving ulcers which later are covered by scabs under which healing takes place. Due to mechanical injury caused by milking and to secondary infections, the lesions heal very slowly. Because of the pain, milking may be incomplete and invasion of germs often gives rise to mastitis, which is one of the important sequelae, the percentage varying according to the preventive and hygienic measures taken.

The most important economic feature has been the drop in milk production.

We have never observed any lesions affecting the feet.

Dr. Strozzi is assistant director of the Instituto Nacional Antiaftosa del Peru; Dr. Ramos-Saco is dean of the veterinary school, University of San Marcos, Lima.

CLINICAL DIAGNOSIS

Before the etiology was determined, this disease was confused clinically with cowpox and only when mouth lesions were observed in a few animals was vesicular stomatitis or foot-and-mouth disease suspected. The exact nature of the disease was determined by means of the serological diagnosis.

From the clinical standpoint, this disease resembles cowpox except that mouth lesions are not described in the latter. During this outbreak, there had also been cases of cowpox as a consequence of pox vaccination of the human population in the region.

It is practically impossible to differentiate, by clinical examination, teat lesions due to cowpox and those due to vesicular stomatitis. The characteristics of umbilicated vesicles, which are usually described as being typical of cowpox, and their extension to other regions of the udder besides the teats, have not been observed by us. Clinically, this condition differs from foot-and-mouth disease by spreading more slowly and having a more limited morbidity. Besides, we do not know of any description of cases of foot-and-mouth disease with primary lesions in the teats.

SEROLOGICAL DIAGNOSIS

The presence of any vesicular type of disease in cattle requires the prompt and precise identification of the true nature of the etiological agent.

Although the biological method suggested by Traum[4] for differentiating vesicular stomatitis from foot-and-mouth disease, based on the inoculation of cattle and horses with the virus, has considerable value, it has been replaced with advantage by the complement-fixation test in recent years. This latter method is effective in the differential diagnosis and the detection of strains of virus and is characterized by its accuracy, rapidity, and low cost.

During the outbreak of this atypical stomatitis, we had the opportunity to isolate some cowpox virus strains by means of the biological method.

The samples taken from the field were sent to the laboratories in glycerin phosphate solution with pH 7.6. In most cases, the material collected consisted of the walls of vesicles taken from the teats and, when possible, the walls of the tongue vesicles were collected.

The serological investigations have been carried on using, directly, the material kept in glycerinated buffer solution according to the technique for the complement-fixation test of Ubertini,[8] with only the modification suggested by Camargo, Eichhorn, Levine, and Tellez Giron[1] for the preparation of the antigen.

Such modification entails the use of sulfuric ether in the purification of the antigen, which allows the action over material preserved in the glycerinated solution without the reaction being disturbed by the anticomplementary activity of the material to be examined.

Table 1 summarizes the results obtained from several serological tests on samples from different farms.

Samples 3, 4, and 6 did not react with any of the hyperimmune serums and when the biological examination was made they proved to be cowpox virus.

TABLE 1—Results Obtained from Serological tests

Origin of Samples		Hyperimmune antiserums				
No. Place	Farm	O	A	C	Ind.	N. J.
1 Ate	M.R.G.					++++
2 Callao	A		++++			
3 Callao*	S.J.					
4 Callao*	Maranga					
5 Chorrillos	E.M.		++++			
6 Chosica*	H.					
7 Chosica	H.					++++
8 Chosica	S.J.					++++
9 Chosica	I.					++++
10 Chosica	S.C.					++++
11 Chosica	M.					++++
12 Chosica	S.A.					++++
13 Chosica	S.C.					++++
14 Chosica	Nana					++++
15	K.					++++
16	P.					++++
17 Collique	H.N.					++++
18 Huacho	S.F.					++++
19 Huaral	C.					++++
20 Huaral	H.					++++
21 Infantas	Sen. S.	++++				
22 S.Augustin						++++
23 Pachacamac	C.					++++
24 Reyes	Repart.	++++				
25 27 Octubre	B.					++++
26 Sayan	A.H.					++++

Table 1 indicates that most of the samples collected during the period of the outbreak belong to the New Jersey type of vesicular stomatitis virus and, also, that there have been outbreaks of foot-and-mouth disease caused by type O and type A viruses.

STUDY OF THE PATHOGENIC ACTIVITY OF THE VIRUS

Since the serological examination of samples from different origins showed that all of them belonged to the same strain of virus (New Jersey), we studied the pathogenic power of sample 20 which originated from Huaral, farm H.

The inoculation material was prepared with vesicle walls obtained from the teats of cows, ground in a quartz mortar, suspended in a buffer isotonic solution of pH 7.6, centrifuged, and made at a concentration of 2 per cent of inoculum per base. Test animals were inoculated with this material.

The inoculation technique is indicated below.

Equine Test.—A total of 10 horses was used. They were divided into two groups: the first group of 2 animals was inoculated intradermally in the tongue; the second group of 8 animals, intramuscularly with 2 cc. of virus suspension.

Thirty-six hours after inoculation, the horses in the first group showed typical symptoms of vesicular stomatitis: temperature 40.5 to 41.0 C., salivation, inappetence, and numerous vesicles on the tongue at the point of inoculation. Forty-eight hours later, the vesicles ruptured leaving extensive eroded areas which healed slowly in ten to twelve days. Secondary vesicular eruptions were not noticed.

The group of animals inoculated intramuscularly did not show any clinical manifestation of the disease. The general condition did not change and the temperature did not show any modification during the fifteen days of observation.

Fifteen days after the inoculation, horses in this last group were experimentally infected in the tongue with a 2 per cent suspension of virus in a phosphated isotonic solution, pH 7.6. None of these animals presented any clinical manifestation of the disease after the inoculation.

Bovine Test.—In this experiment, 10 cows were divided into two groups and inoculated in the same manner as the horses except that a 4-cc. dose of virus was used for the intramuscular inoculation. Both groups reacted in a manner identical with that of the horses.

The group injected by the intramuscular route was also inoculated fifteen days later in the tongue and were completely resistent to this inoculation.

Guinea Pig Test.—Ten guinea pigs were inoculated in the foot pads. Thirty-six hours later, all the inoculated animals showed primary vesicles at the inoculation point and vicinity. In this first passage of the virus, secondary lesions which would show generalization of the virus were not seen.

The behavior of the virus inoculated in all the different species of animals mentioned is typical of the vesicular stomatitis virus.

The tests already mentioned also show that in the equine and bovine species, when the virus is inoculated intramuscularly, it does not cause disease but confers a solid immunity against a subsequent virus inoculation intradermally in the tongue.

SANITARY MEASURES

As the disease was spreading and causing economic losses, it was decided to induce artificial resistance in susceptible animals by vaccination. A vaccine was prepared, with the virus isolated from the field, and used on uninfected farms in those regions where the disease was threatening. The virus used in the vaccine was adsorbed by aluminum hydroxide. The technique used in the preparation of this vaccine and the results obtained will be published in another report.

The disease spreads rather slowly in a herd, so having proved that the virus inoculated intramuscularly does not reproduce the disease but confers immunity, we inoculated all apparently healthy animals in herds already infected. The virus used in the preparation of the inoculum was always derived from the herd in which it was used. The virus suspension used in the inoculum was prepared by grinding finely the walls of the mammary vesicles and suspending them in a phosphated isotonic solution so as to obtain a 1 per cent virus concentration. The suspension was then centrifuged or filtered through paper and inoculated intramuscularly, 1 cc. per animal. New cases continued to develop for fifteen days following inoculation, but not after that.

On two farms, one of which was already infected, where the animals had been vaccinated with the type of vaccine mentioned, an outbreak of vesicular type of disease affecting the teats was observed a month and a half following inoculation in 1 case and after vaccination in the other. Serological tests excluded vesicular stomatitis as the etiological factor in the disease, but animal inoculation proved it to be caused by cowpox virus.

SUMMARY

In this work, an atypical form of vesicular stomatitis is described in dairy cattle, with lesions localized primarily in the teats.

Reference is made to the possibility of milking being an important factor in spreading the virus within a herd.

The complement-fixation test has shown the New Jersey strain of the vesicular stomatitis virus to be the cause of this disease.

This atypical form of vesicular stomatitis shows differential clinical characteristics from foot-and-mouth disease but not cowpox. The serological test and animal inoculation of the virus are the only reliable means of determining precisely the etiology of the disease.

Uninfected herds that had been exposed were vaccinated with the New Jersey strain of vesicular stomatitis virus adsorbed by aluminum hydroxide. The technique of the preparation of this vaccine and the results obtained will be published later.

Apparently healthy animals of infected herds were inoculated intramuscularly with virus isolated from the same herd. New cases of diseased animals were observed up to fifteen days following the inoculation, but not after that.

The vesicular stomatitis outbreak here described has spread over an extensive area and has caused serious economic damage which contradicts what is usually stated in regard to this disease.

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Cat Scratch Disease Not New

In 1950, a clinical entity characterized by subacute lymphadenitis, in which the lesion frequently suppurated, was described. The patient had usually been scratched by a cat. A similar clinical entity had been recognized, but was not reported in the United States, about twenty years earlier. However, in 1889 what now seems to be a similar condition was described in France as "Parinaud's oculoglandular syndrome."

Three cases of conjunctivitis accompanied by enlargement of the regional lymph nodes had occurred in slaughterhouse workers. Eight similar cases were described in 1950, 7 of which had been exposed to cats. Just recently, 4 similar cases were negative to tests for tularemia, lymphogranuloma venereum, and syphilis but were strongly positive to the antigen of cat scratch fever. Leptothrix, which had formerly been held responsible for many such cases, probably was present just as a saprophyte. Furthermore, this condition is apparently not transmitted from man to man. It has clinical features similar to cat scratch fever except for the primary location. The location apparently varies with the portal of entry, which in Paranaud's syndrome is probably the conjunctiva.—*J. Am. M.A., Aug. 29, 1953.*

Parasites and Periodic Ophthalmia.—In Germany, 19 horses with periodic ophthalmia were autopsied. In 18, the adult *Onchocerca reticulata* were found in the ligamentum nuchae. In 14, the larval form of the parasite was demonstrated in eye tissues, particularly the cornea, sclera, and conjunctiva. In 6 normal horses, the microfilariae could not be demonstrated.—*Vet. Bull., June, 1953.*

Brucella Lesions in Guinea Pigs

In guinea pigs *Brucella suis* infection produced marked nodular changes in the spleen, testicles, and liver with a tendency to suppuration and giant-cell accumulations. The lesions produced by *Brucella melitensis* are less marked while *Brucella abortus* produces only enlargement of the spleen and grayish, miliary foci in the liver.—*Vet. Bull., Aug., 1953.*

The Transmission of Proliferative Stomatitis of Cattle

CARL OLSON, Jr., D.V.M., Ph. D., and TADAS PALIONIS, D.V.M.

Lincoln, Nebraska

UNDER THE term "proliferative stomatitis and esophagitis (x disease)," Udall[17] gave a brief description of what has later been called hyperkeratosis (x disease) of cattle[10] or bovine hyperkeratosis.[12] The two principal unequivocal manifestations of bovine hyperkeratosis have been hyperkeratosis of the skin and lesions of the oral cavity. Although the mouth lesions have been called oral papillomas[12] because of their proliferative cauliflower-like appearance, they are perhaps more correctly described by the term "proliferative stomatitis" which will be used in this report.

Before it was known that the hyperkeratosis of the skin was due to toxic factors,[1-3,9,11,12,15,18,19] various experiments had been done in attempts to transmit bovine hyperkeratosis from one animal to another. In some of these attempts, lesions developed on the oral mucosa following inoculation with material from oral lesions from a case of the spontaneous disease. The literature on these reports has been reviewed elsewhere,[13] (also see Discussion) and will not be repeated here. It is sufficient to say that the results were not uniform; neither was a specific agent associated with the condition nor were serial passages of the disease accomplished.

Observations on field as well as experimental cases of bovine hyperkeratosis indicate that a proliferative stomatitis is not always associated with the disease. It is more often seen in the earlier stages of the disease and may last for variable lengths of time.[10,12,13] These observations become understandable since information presented in this report indicates that proliferative stomatitis is due to a virus-like agent which is without effect in an older calf on normal feed but will produce stomatitis in a calf that has been receiving a hyperkeratosis-producing substance. It was also found that very young calves were susceptible without special treatment.

From the Department of Animal Pathology and Hygiene, University of Nebraska, Lincoln.

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MATERIALS AND METHODS

Most of the test calves were 3 to 6 months old, of grade dairy stock. They were obtained from farms where there was no history of bovine hyperkeratosis. The test calves were fed prairie hay supplemented with oats, cracked corn, and either soybean or cottonseed meal. The hyperkeratosis-producing feedstuff was pellets composed of dehydrated alfalfa alone or dehydrated alfalfa plus 2.5 per cent dicalcium phosphate which had been found capable of causing bovine hyperkeratosis.[11] Usually these pellets were fed five to seven days prior to exposure with proliferative stomatitis material and for about seven days subsequent to inoculation. If the calves did not eat their daily ration (¼ to 1 lb.) of pellets, it was administered by a stomach tube.

The calves were housed in individual pens. The exposed animals were kept in a separate building and cared for by a separate attendant in order to avoid transfer of the infection to other animals.

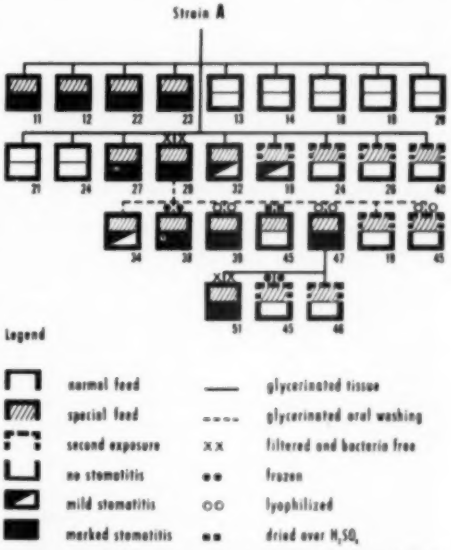


Fig. 1—A diagram showing transmission experiments with strain A proliferative stomatitis agent through three serial passages. The legend gives explanatory detail of materials used for inoculation and also the results obtained.

Proliferative stomatitis material was obtained from two sources. Strain A was obtained from a calf with extensive mouth lesions (fig. 2) which had developed during a field outbreak of bovine hyperkeratosis in Pennsylvania.* Strain B material was obtained from a field case of bovine hyperkeratosis occurring on a farm in Nebraska. The materials were stored in 50 per cent glycerin with physiological saline solution. Inoculums were prepared by grinding the glycerinated proliferative stomatitis tissue in physiological saline solution at a dilution of about 1:10. Penicillin was added at the rate of about 4,000 units per milliliter of suspension. The supernatant fluid was injected with a small needle into and under the epithelium of the lower lip of the test calves. Usually, two to four sites were used. In a few instances, inoculation was made by scarifying the oral epithelium with a stiff brush or sandpaper and spraying or swabbing the inoculum onto the surface.

Material for serial passage was obtained either by biopsy or by spraying physiological saline solution onto the surface of the lesion and catching the fluid as it dripped from the mouth. These materials were either stored in 50 per cent glycerin, at low temperatures (-35 or -60 C.), or lyophilized.

Filtration experiments were conducted using either a Seitz asbestos filter or a porcelain filter (Selas Corp., Philadelphia, Pa.). During filtration, the possibility of leakage was tested by addition of a bacterial culture to the material prior to filtration. Sterility of the filtrate indicated no leakage.

RESULTS

Proliferative stomatitis material when inoculated into the oral cavity of calves 3 months of age or older, which had not had preparatory feeding with hyperkeratosis-producing pellets, failed to produce stomatitis (fig. 1, calves 13, 14, 18, 19, 20, 21, 24). Four young calves kept at the laboratory and inoculated at 3, 9, 14, and 30 days of age developed stomatitis although they were not fed the pellets. Five calves, 4 to 61 days old when inoculated, and nursed by cows on pasture, were also susceptible. Similar material inoculated into the oral cavity of older calves which were receiving the pellets caused a proliferative stomatitis (fig. 1, calves 11, 12, 22, 23, 27, 29, 32).

The first evidence of the reaction was swelling and congestion at the site of subepithelial inoculation. This was usually evident five to ten days after inoculation and was followed by ulceration in these areas from seven to fifteen days after inoculation. The ulceration lasted from a week to two weeks and some times was accompanied by a proliferation of epithelium (fig. 3). When this proliferation occurred, it developed two to three weeks after inoculation and lasted as long as three weeks in some animals. About the time ulceration developed at the inoculation site, there were small congested areas scattered elsewhere on the mucosa of the mouth. Often the lesions spread to the lips, gums adjacent to incisors, buccal mucosa, and mucosa of the tongue and palate. Frequently, these would become ulcerated and sometimes developed a proliferative reaction (fig. 4, 7). Two to three weeks after inoculation, red areas were often noted on the outside of the lips, muzzle, and

*We are indebted to Dr. A. L. Bortree, Pennsylvania State College, State College, Pa., for this material.

Legends for Figures on Opposite Page

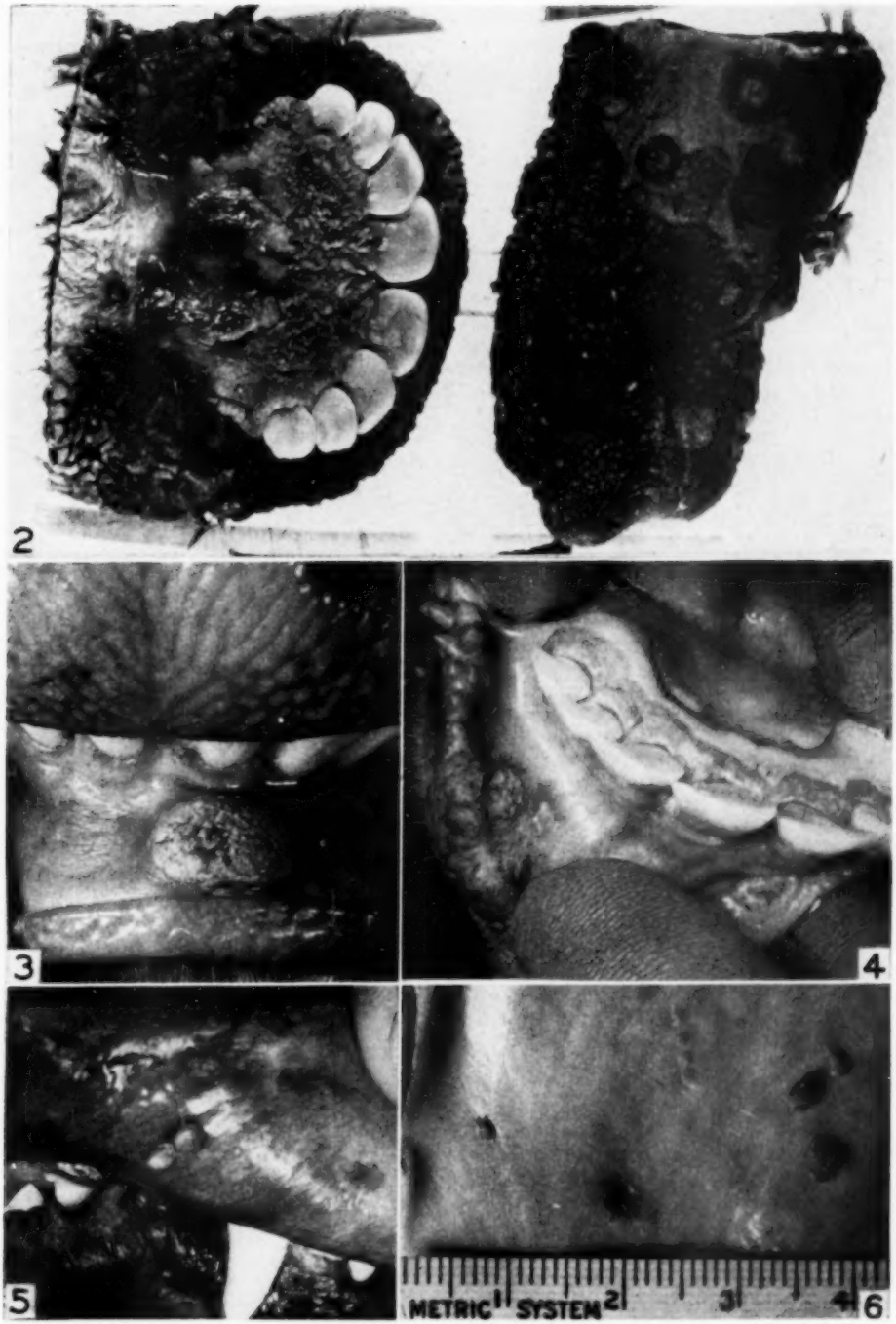
Fig. 2—Section of lower jaw and tip of tongue showing marked proliferative stomatitis. Strain A originated from this case. The mucosa of pharynx and epiglottis was also involved. There was marked ulceration of mucosa in the upper half of the esophagus. Histologically, these lesions show marked papillary proliferation of epithelium with "balloon" vacuolar degeneration of deeper layers of epithelial cells, some of which contained "inclusion bodies." Necrosis and ulceration with bacterial infiltration was frequent near the surface of the lesions. There was a polyblastic inflammatory reaction in the submucosa.

Fig. 3—Lesion inside lower lip of calf 51 showing the proliferative reaction at the site of inoculation sixteen days after exposure to filtered proliferative stomatitis material. Note the circumscribed lesion and its raised proliferative character which could be regarded as papillomatous.

Fig. 4—Calf 22 eighteen days after exposure to proliferative stomatitis material. The point of inoculation was at the edge of the lower lip immediately above the end of the gloved finger. Here may be noted a slight proliferative reaction which is also evident behind the incisors and along the edge of the lip.

Fig. 5—Two papilloma-like projections of the buccal mucosa of a dog at the site of inoculation with proliferative stomatitis material. This picture was taken seventeen days after inoculation. The lesions were removed one day later and did not recur.

Fig. 6—Mucosa of esophagus from cervical region of a calf that died with bovine hyperkeratosis. Five larger, irregular-sized, papilloma-like lesions are evident. There are four smaller papilloma-like lesions arranged in a row.



—*Photograph and description, courtesy of Dr. A. L. Bertree*

nostrils; the epithelium would become necrotic and the lesions would last for one to four weeks before healing. In some animals, the stomatitis would become so troublesome that the calves would not eat well.

There was no evidence of an elevation of body temperature during the time stomatitis was developing and existed. Most of the calves received an insufficient quantity of the pellets to cause actual hyperkeratosis of the skin. The proliferative stomatitis which developed in these calves was similar in appearance and character to that which occurred in those calves receiving a quantity of pellets sufficient to cause actual hyperkeratosis. The only difference seemed to be in the duration of the stomatitis which was longer in those calves receiving the larger amount of hyperkeratosis-producing pellets.

Most of the older calves were inoculated with the proliferative stomatitis material during the time they were receiving the pellets. One calf (calf 32, fig. 1) received 20 lb. of hyperkeratosis-producing pellets over a period of thirty-three days. This was not sufficient to cause hyperkeratosis of the skin but evidence of poisoning was manifest by loss of weight and a decrease of blood plasma values for carotene and vitamin A. Calf 32 was inoculated with stomatitis material fifty-four days after the pellets were fed. A mild stomatitis followed seven days after exposure and lasted for twenty days.

Proliferative stomatitis material obtained from biopsy remained active for seven months when kept in 50 per cent glycerin at about 4 C. The activity was retained after filtration through a Seitz filter (fig. 1, calf 29), and after filtration through a porcelain filter (fig. 1, calf 51). In both instances the filtrate was free of bacteria. An oral washing of lesions of proliferative stomatitis remained active for ninety-four days when stored in equal parts of glycerin and kept at about 4 C. The same oral washings were lyophilized and the material was active when resuspended and inoculated into calves one year later. The same oral washing material was frozen at –35 C. and stored for seventy days without losing its activity (fig. 1, calf 39).

One calf (fig. 1, calf 19) did not develop stomatitis when it was first exposed. It was not receiving hyperkeratosis-producing pellets at the time of the initial exposure. Thirty-five days later when it was receiving the pellets, it was reinoculated and developed a mild but definite stomatitis. It was exposed a third time, forty-nine days after its second exposure, with material active in other calves (fig. 1, calves 34, 38, 39, and 47) and failed to react to the third exposure. Another calf (fig. 1, calf 24) was not fed pellets at the time of the first exposure but was fed pellets at the time of the second exposure 120 days later. No stomatitis occurred following either the first or second exposure.

Various attempts were made to test the susceptibility of the calves to reinoculation. These included cross-immunity tests between strains A and B as well as reinoculation with the same strain. Some calves which had developed stomatitis following inoculation with strain A were subsequently exposed to strain B and vice versa. In all instances, they were refractory to the second inoculation (for example, fig. 1, calf 46 had stomatitis produced with strain B 58 days previous to its second exposure with strain A).

Contact exposure of 3 calves receiving hyperkeratosis-producing pellets with other calves that had active proliferative stomatitis caused the development of stomatitis in the 3 calves. Material from 1 of the 3 calves was used to transfer the disease to experimental calves. Typical proliferative stomatitis developed. Some of the material was inoculated into a calf which 110 days previously had stomatitis from strain A material. The calf was refractory to the second exposure. One of the contact-exposed calves was inoculated with strain A material after its initial stomatitis had subsided. It was refractory to the second exposure (fig. 1, calf 40).

During the course of these experiments, localized papular lesions developed on the skin of the hands of two people who had been working with affected calves. In one person, a single lesion developed first as a reddish area. This slowly enlarged so that in three weeks it was about 8 mm. in diameter, consisting of a central depressed white area with a violet periphery. The superficial layers of epithelium came off, leaving a surface covered with serum. Healing gradually took place in three weeks. The second person developed a similar lesion on the back of the hand several months

Fig. 7—Calf 23 eighteen days after inoculation with proliferative stomatitis material. Ulcerative and proliferative areas can be seen on the mucosa of the ventrolateral aspect of the tongue. This area was adjacent to the inoculation site at the right corner of the lip.

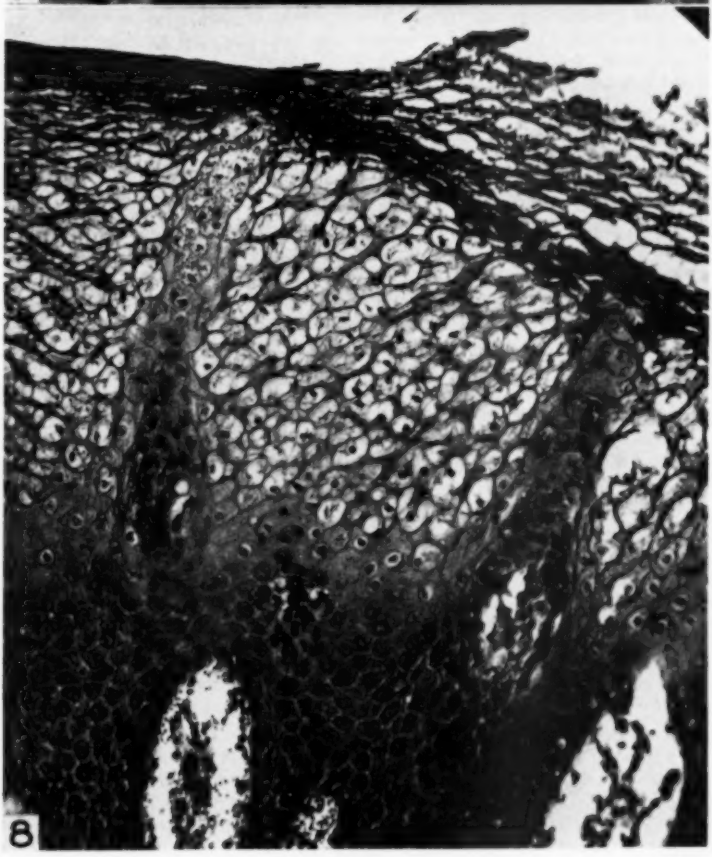


Fig. 8—Epidermis at edge of slightly swollen site where proliferative stomatitis agent had been inoculated five days previously. Notice the swelling ("ballooning") of cells in stratum granulosum which was a rather constant histological feature. x 200.

later. The natural course of this lesion was interrupted by biopsy, but several months later another lesion developed on the finger. This began as a 3-mm. papule with a thin, irregular, pearly line giving a sharp border. Seven days later, the violet-colored lesion was about 5 mm. in diameter with a 1-mm. white center. Biopsy was done at this time and a portion preserved in 50 per cent glycerin. The glycerinated material was subsequently inoculated into the oral mucosa of test calves fed the pellets. Both developed a mild stomatitis. One of them was exposed to strain A material sixty-three days later and was refractory (fig. 1, calf 26).

Strain A material was used to inoculate other species of animals. Two sheep, 2 pigs, 2 rabbits, and 2 guinea pigs were fed hyperkeratosis-producing pellets previous to inoculation into oral mucosa. All of these animals remained negative. Two horses were similarly exposed but were not fed the pellets. These animals remained negative. Two dogs, 6 months old, were inoculated in the oral cavity. They were not fed pellets. Proliferative growth was observed at both sites of inoculation in 1 dog thirteen days later (fig. 5). Eighteen days after inoculation, the papillomas were excised. There was no recurrence. Four more dogs were inoculated with proliferative stomatitis material, 2 of which received the pellets. These 4 dogs remained negative.

The possibility of a relation between the common oral papilloma of the dog and proliferative stomatitis was investigated. Two of the dogs refractory to proliferative stomatitis material were later found susceptible to canine oral papillomatosis material. A calf fed hyperkeratosis-producing pellets was inoculated in the mouth with canine oral papilloma material. It remained negative and was later found susceptible to proliferative stomatitis material (fig. 1, calf 38).

DISCUSSION

The results seem to indicate rather clearly that proliferative stomatitis can be readily transmitted from one calf to another provided the calf is prepared by the feeding of hyperkeratosis-producing feedstuff. In addition to using hyperkeratosis-producing pellets to predispose to inoculation, 1, 4-month-old calf was found susceptible after administration of pentachlorinated naphthalene. In general, proliferative stomatitis did not develop when hyperkeratosis-producing material was not fed prior to, or in conjunction with, exposure of the test calves, but exceptions to this were young calves (3 to 30 days old) which were susceptible without this feedstuff.

The histology of the experimentally produced lesions was essentially similar to natural proliferative stomatitis (fig. 8). Generally, the degree of proliferation in the test animals was somewhat milder than in the natural disease. There were some variations, depending upon the stage of the disease.

These results are not entirely in harmony with the observations of others. Inoculation onto the scarified oral mucosa with mouth lesion material from cases of bovine hyperkeratosis was reported (Olafson and Fincher, cited by Hagan[6]) to cause the development of wartlike lesions in the mouth of test calves. Gibbons[5] stated that rubbing exudate from lesions of the mouth onto the scarified tongue of a calf would sometimes produce a local reaction. Jensen[8] inoculated 2, 4-month-old calves, which had been kept on a ration very low in vitamin A, with ground bovine oral papilloma material. In addition to exposure by injection and scarification of oral mucosa, each calf received, intravenously, 10 ml. of whole blood from the same donor. Ten days later, there were well-defined papillomas (5 mm. in diameter and 1 mm. high) under the tongues of both calves. An exact comparison of the observations by other workers is difficult since there was no indication of how regularly the condition could be produced or whether it could be carried in serial passage.

Sippel[16] received from Pennsylvania some of the same material from which our strain A was developed. He prepared a suspension of affected mucosa from the palate and nostrils and inoculated a calf intradermally, on scarified skin, on the gum, orally, and intranasally. No systemic symptoms developed in the calf. A transient swelling developed at the site of the inoculation on the gum. A proliferative lesion developed in ten days where the skin had been scratched. It dropped off in pieces, like a scab, between sixty and seventy-five days after inoculation. This result could not be duplicated. These lesions were somewhat different from those observed in our study.

Bortree[4] brought 3 field cases of bovine hyperkeratosis with proliferative stomatitis (one of them was the source of strain A, *see* fig. 1) into a barn housing 10 other experimental calves aged 6 months to 1½ years. Three weeks later, various lesions developed in the mouths of 10 calves, apparently as a result of contagion from the 3 field cases. The lesions varied from white macules about the muzzle and nostrils to inflammation of buccal mucosa with ulceration and, in some cases, proliferation of the epithelium. Four of the 10 calves were on normal feed and the rest were on rations being tested for toxicity, or known to be able to produce hyperkeratosis. The rations being tested contained one of the following: rancid lard, naphthalene, pentachlorophenol, a German wood preservative, extract of a feed concentrate, or toxic hay. Three other calves in another part of the barn did not develop mouth lesions. It would seem from this experiment that proliferative stomatitis was contagious and that the agent in its natural state was capable of causing oral lesions of short duration even in calves on a normal feed. Our failure to infect older calves on normal feed with material from the same source may have been due to partial inactivation of the agent from storage in glycerin or other unknown factors.

Previous to the development of these two strains of proliferative stomatitis, we made numerous attempts to produce the condition. Oral papillomatous material from field cases of bovine hyperkeratosis was prepared while fresh and inoculated onto the oral mucosa of test calves on "normal" rations. Ulcerative lesions would develop in a day or two. These lesions did not develop into the characteristic proliferative stomatitis. The ulcerative lesion would not develop if the inoculum was treated with an antibiotic prior to injection.

Proliferative lesions have been observed in the esophagus at necropsy of cases of hyperkeratosis, but we were not able to examine the esophagus of our living experimental animals. In one fatal case of experimentally produced bovine hyperkeratosis, some proliferative papilloma-like lesions were found in the esophagus at necropsy. In one area, there was a row of small papillomas, arranged as though they had resulted from injury with the sharp point of a stem of hay or similar material (fig. 6). Possibly such lesions arise as result of injury to the epithelium which permits introduction of the virus.

It would appear that the agent of proliferative stomatitis of cattle was capable of causing lesions on the skin of man. Three such granulomatous lesions developed during the course of our work. In one instance, the lesion was used to produce the experimental disease in calves. One of these calves was immune to a subsequent exposure with a proliferative stomatitis material from a calf, thus indicating immunological identity of the agent responsible for the two conditions.

The papilloma-like condition which developed in 1 dog exposed to proliferative stomatitis material from a calf is difficult to understand. Repeated attempts to duplicate this result were negative. The proliferative stomatitis agent was not immunologically related to the agent of oral papillomatosis of the dog, since dogs exposed to the proliferative stomatitis material were subsequently susceptible to oral papillomatosis of the dog. Further evidence of a difference between these two agents is suggested by the fact that a calf was refractory to oral papillomatosis material of the dog, but when later inoculated with proliferative stomatitis material it developed typical lesions.

Some calves seem to be naturally resistant to the agent of proliferative stomatitis even though they are given a preliminary feeding of hyperkeratosis-producing feedstuff. Two examples of this type of resistance were calves 24 and 45 (fig. 1). Calf 24 was exposed twice to active proliferative stomatitis material. Hyperkeratosis-producing feedstuff was given at the time of the second exposure, but calf 24 did not develop proliferative stomatitis as result of the second exposure as did calf 19 (fig. 1) which had been similarly treated. Calf 45 was used three times (fig. 1) and each time it received hyperkeratosis-producing feedstuff at the time of exposure. The first exposure was with material which had dried and the agent may have been inactivated. The second exposure was with material frozen and stored at −60 C. The third inoculation was with lyophilized material but the calf remained refractory.

The possible relationship of the agent of proliferative stomatitis to the agent of cutaneous papilloma of cattle has been con-

sidered. The agent of proliferative stomatitis was inoculated onto the skin of 4 calves simultaneously with exposure of the oral cavity. Two of the calves received hyperkeratosis-producing pellets and 2 did not. In no instance was there any characteristic reaction at these sites of inoculation. These 4 calves were also inoculated at the same time with material containing the agent of cutaneous bovine papilloma and 1 of them developed typical cutaneous papilloma.

In other experiments, one strain of bovine cutaneous papilloma material was inoculated onto both the skin and oral mucosa of 6 test calves. Papillomas developed only on the skin of 2 calves. Although these calves were not fed hyperkeratosis-producing feedstuff, the results indicate lack of activity of the bovine cutaneous papilloma agent on oral mucosa of animals whose skin was susceptible to the same inoculum. Proliferative stomatitis materials of both strains A and B were injected into the skin of horses susceptible to bovine cutaneous papilloma agent and there was no reaction. The bovine cutaneous papilloma agent will provoke a reaction in the skin of the horse.[14]

A possible relation between the agent of proliferative stomatitis and that of vesicular stomatitis deserves consideration. Hanson's[7] review of vesicular stomatitis indicates that the horse and pig will develop vesicular stomatitis. Both horses and pigs were refractory to proliferative stomatitis.

SUMMARY

Transmission experiments with proliferative stomatitis in calves indicate that this is an infectious and contagious disease caused by a filterable agent. Preliminary or simultaneous feeding with hyperkeratosis-producing feedstuff was necessary to make older animals receptive to action of the agent. Very young calves were susceptible without preliminary treatment. Immunity follows an active attack of the disease. The agent of proliferative stomatitis is capable of causing a chronic lesion on the skin of man. The agent appears to be different from that of cutaneous papillomatosis of cattle, oral papillomatosis of the dog, and vesicular stomatitis.

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Teschen Disease in Germany.—In Germany, Teschen disease is controlled by the slaughter of all pigs on infected premises and rigorous disinfection. The use of vaccines and serums against Teschen disease is prohibited.—*Vet. Bull., June, 1953.*

Bovine Hyperkeratosis (X Disease)

R. P. LINK, D.V.M., Ph.D.

Urbana, Illinois

IN 1941, a new and peculiar disease of cattle was observed and reported in New York state.[6] It was a distinct entity not reported previously. Since the cause was unknown, the term x disease was applied. The term "hyperkeratosis" is now used to designate the disease because of the characteristic tissue changes. Olafson[10] described the symptoms, lesions, and histopathology of the disease in 1947.

A survey conducted by the Bureau of Animal Industry[20] in 1948 revealed the widespread incidence of the disease. Investigations were inaugurated at several experiment stations to study causative factors and methods of control.

Losses from bovine hyperkeratosis are sporadic and may involve only a few animals or an entire herd. One conservative estimate places the value of animals that have died or were destroyed because of this malady in the southeastern part of the United States in excess of $4,000,000. In Tennessee, animals valued at more than $1,000,000 have been lost due to this disease. Losses in Wisconsin during 1952 were estimated to exceed those in Tennessee. The disease was known to occur on 42 farms in Illinois during 1952. Mortality varied from 4 to 87 per cent in these herds. Outbreaks of this disease have been reported from several states during the early part of 1953, and although accurate figures on the number of animals affected are not available, one estimate placed the loss in excess of $20,000,000. Mortality is not an accurate estimation of economic loss, for many calves which recover are stunted and mature animals may not produce or gain in weight as expected. Although tissue changes occur in some of the reproductive organs, the report of Olson *et al.*[14] indicated no impairment of reproductive ability in heifers which had recovered. Some preliminary observations suggest that bulls which have recovered are fertile and have normal breeding ability.

Calves are most often and most severely affected, but cattle of all ages are susceptible. The disease is characterized by lacrimation, salivation, nasal discharge, variable appetite, depression, intermittent diarrhea, red swollen areas in the mouth and on the muzzle, polyuria, and loss of weight. Thickening and wrinkling of the skin, with or without loss of hair, may occur on the neck and withers and in some cases may extend over most of the body. Internal lesions include: cystic dilatation of renal tubules; squamous metaplasia of epididymes, seminal vesicles, Gartner's ducts, and salivary glands; mucoid papillary proliferations in large bile ducts and gall bladder; thickening of the gall bladder; fibrosis in pancreas and kidney; and proliferation of small bile ducts in the liver. Papillary growths may be found in the mouth and esophagus. Data presented by Olson and Palionis[16] suggest that these lesions are caused by an infectious agent and are not necessarily found in all cases of hyperkeratosis.

The work of Olafson,[10] Gibbons,[4,5] and Olson *et al.*[12] demonstrated that an infectious agent was not the cause of the disease. Repeated attempts to transmit the disease using blood, tissues, and secretions from affected animals as inoculum were without success. Contact experiments have also given negative results.

Evidence that bovine hyperkeratosis is caused by a toxic substance or substances is presented in several reports. The presence of a probable hyperkeratogenic substance in feed was reported by Olson *et al.* in 1950.[12] Olson and Cook[13] produced the disease by feeding pelleted dehydrated alfalfa and dehydrated alfalfa plus dicalcium phosphate which they suspected as being the cause of an outbreak of the disease.

Olafson and McEntee[11] were able to produce the disease by feeding a processed wheat concentrate. McEntee *et al.*[8] proceeded with fractionation of an ether extract prepared from the processed concentrate and reported the causative substance was present in the free fatty acid fraction but not in the phenolic and neutral steroid fractions nor in the ether extract residue. Later work by Hansel *et al.*[7] demonstrated

From the College of Veterinary Medicine, University of Illinois, Urbana.

that the toxic substance was not a fatty acid. The similarity of some of the lesions of hyperkeratosis to those seen in vitamin A deficiency prompted them to study the effect the hyperkeratosis-producing substance exerted on vitamin A metabolism. They found that plasma vitamin A values decreased rapidly in calves which were developing hyperkeratosis.

Wagener[21] found that a wood preservative caused hyperkeratosis in cattle in Germany. He was able to produce the disease experimentally by housing cattle in a cabin which was painted with the wood preservative. A quantity of this wood preservative was sent to the United States for study by research workers at Cornell University and Pennsylvania State College. They also produced hyperkeratosis in cattle by using this wood preservative which is known to contain chlorinated naphthalene.

Miller *et al.*[9] found that a certain cutting of timothy hay would produce the disease when fed to susceptible animals. This hay may have been contaminated by a wood preservative. They also found evidence that the disease was caused by continued exposure of calves to wood which had been repeatedly saturated with a disinfectant of coal tar origin. Washko *et al.*[22] reported observations in which the disease may have been caused by cattle being exposed to boards treated with a creosote-like product. Certain petroleum products were found by Bell[1] to be hyperkeratogenic. He produced the disease in calves by the oral administration of a small amount of a lubricant. Later, Bell[2] working with the manufacturer of the lubricant found the toxic substance was a chemical additive (a highly chlorinated naphthalene compound) used as an extreme pressure agent in the manufacture of the lubricant.

Sikes *et al.*[10] demonstrated that typical symptoms of this disease could be produced in cattle by feeding highly chlorinated naphthalenes or lubricants containing them or by applying such lubricants to the skin.

Studies at Cornell and Tennessee indicated that the causative substance is transmitted through cow's milk. Calves nursed on cows receiving the disease-producing substance developed typical symptoms of the disease. This occurred in the Tennessee study even when the agent was applied to the skin along the back of the cow.

Bell's[3] recent report indicates that a small amount of highly chlorinated naphthalene is capable of producing the disease in calves. He also found that the degree of chlorination of naphthalenes influenced their hyperkeratogenic activity, but that di- and trichloronaphthalenes did not produce hyperkeratosis in calves when used under the conditions of his study.

Olson[15] has reported a milk factor responsible for hyperkeratosis in calves on one farm. The milk factor appeared to be in the skim milk and not in the cream.

In an experiment supervised by Phillips and O'Connell[17] the disease was produced by feeding a pelleted protein concentrate of the same brand as had been fed on numerous farms in Illinois where the disease appeared. Apparently not all lots of feed produced by this mill contained the hyperkeratogenic substance for it has been fed on many farms where hyperkeratosis did not occur. This suggests the possibility that certain lots of feed may have become contaminated by a hyperkeratogenic substance. Schmidt[18] has stated that the cause of some recent outbreaks of this disease in Texas was chlorinated naphthalene in cottonseed pellets which were fed the cattle. A U.S.D.A. release stated that the machines used in the pelleting process were lubricated with a grease containing highly chlorinated naphthalene. It was found that these machines as greased allowed enough chlorinated naphthalene to go into the pellets to produce hyperkeratosis in cattle when fed in the amounts and over the period of time normally practiced in livestock management. The disease has been reported as occurring in several other southwestern and western states presumably from the same cause.

There is no specific treatment for bovine hyperkeratosis. Vitamin A therapy may be of value if the causative substance is removed and the pathological process has not advanced to an irreversible state.

Tests conducted with the newer insecticides and wood preservatives indicate they are probably not capable of producing the disease. Due to the rather variable chemical composition of some petroleum and coal tar products[23] further work is required to determine their potential hazards. The observation of Miller *et al.*[9] that hyperkeratosis was probably caused by exposure of calves to wood which had been saturated with a disinfectant of coal tar origin suggest that such products may contain a hyperkeratogenic substance.

Domestic supplies of wood preservatives do not contain chlorinated naphthalenes but they are extensively used in foreign wood preservatives.[25] Approximately 20 per cent of the wood preservative used in the United States during 1951 was imported.[24] The increased volume of imported wood preservative used since the war may be one reason for the increased incidence of the disease.

Certain types of gears require lubricants which have greater load-bearing properties and heat resistance than possessed by petroleum lubricants. These deficiencies are corrected by the addition of a small amount of highly chlorinated naphthalene. Some lubricant manufacturers use other compounds to accomplish this objective. Of these, only the highly chlorinated naphthalene additives are known to be hyperkeratogenic.

Some lubricant manufacturers are discontinuing the use of chlorinated naphthalene in lubricants intended for use on farm machinery. However, they continue to be used in many lubricants, particularly the extreme pressure types. Used oil which may contain transmission or differential drainings should be considered a potential source of hyperkeratogenic substances.

Bell's work published in January, 1952, proved that the disease was caused by a lubricant. Application of that information could have prevented some of the losses from hyperkeratosis since that time. The majority of feed-pelleting machines are constructed so that the lubricant works into the pellets. Lubricants used on other machines employed in the harvesting and processing of livestock feeds may also be sources of contamination. Veterinarians should consider all lubricants as possible sources of hyperkeratogenic substances.

There appears to be variation in susceptibility of different species of animals to the toxic action of chlorinated naphthalenes. Several reports indicate that man is susceptible to the toxic effects of these compounds. Since the hyperkeratogenic substance may be present in the milk of lactating cows, these substances may present a hazard to human health.

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Erthromycin is often effective against *Micrococcus pyogenes* (*Staphylococcus aureus*) infections which are resistant to penicillin, streptomycin, aureomycin, and terramycin.® It was highly effective in 6 of 8 patients with staphylococcic septicemia. The other 2 patients died when the organism rapidly became resistant. It was also effective in staphylococcic ileocolitis, meningitis, and throoat and urinary tract infection. No serious toxic reactions to erythromycin were observed.—*J.Am.M.A., Aug. 22, 1953.*

Virus Pneumonia of Swine

Virus pneumonia causes a marked retardation of growth in some pigs but its effect on others is not obvious. Therefore, two experiments, one in summer and the other in winter, were conducted in England on groups of 16 pigs each, 8 being infected and 8 left as controls. Pigs 8 weeks old were used and when each attained a weight of 200 lb., it was slaughtered and the lungs examined. The groups were maintained in similar environments with separate attendants. While no coughing was detected in the control groups, all those infected in the summer were coughing by the nineteenth to twenty-fourth day. Surprisingly, those infected in the winter coughed less than those infected in the summer group.

In the summer, the first control pig reached 200 lb. on the 113th day of the experiment and the last pig on the 133rd day, while the first and last infected pigs reached 200 lb. the 121st and 203rd days respectively. The average growth rate was 1.29 lb. a day for the controls and 1.11 lb. for the infected pigs. The control pigs required 3.39 lb. of food to produce 1 lb. of gain; the infected pigs 4.25 lb. Thus, the infected pigs required 25 per cent more food.

In the winter experiment the growth differences were even more marked, the controls making an average growth of 1.19 lb. per day, the infected group 0.92 lb. per day. Extensive field observation suggests that differences in the rate of growth are frequently much more marked under natural conditions.—*Vet. Rec., Aug. 15, 1953.*

Antibiotics and Guinea Pigs

When terramycin,® chlortetracycline (aureomycin), and chloramphenicol were fed to guinea pigs at the rate of 30 mg. per day for ten days, the intestinal microflora was considerably changed. Terramycin and aureomycin caused a partial or complete disappearance of staphylococci, coliform organisms and, certain others while Proteus, *Streptococcus faecalis*, and other resistant organisms multiplied until the total flora population was approximately doubled. In contrast, chloramphenicol inhibited the growth of the intestinal flora as a whole without selective action.—*Vet. Bull., Aug., 1953.*

Effects of Vacuum Level and Milking Duration on Normal Udders

When mechanical milkers are associated with an increase in mastitis, the factors to be considered are excessive vacuum, leaving the machine attached too long, and attaching it before adequate let-down has occurred. To test the effect of these factors, first-calf heifers (3 Jerseys and 9 Holstein-Friesians) were arranged in replicates of 3. One in each group was milked with a vacuum of 10 in. of mercury, 1 at 13 in., and 1 at 17 in. The two halves of each udder were milked differently—on one half, the machine was left attached a normal period while on the other half it was left twice as long. The effects were judged by the leukocyte count of the milk, the chloride value, the PH value, the mastitis organism count, and the physical condition of the quarter. At the end of forty weeks there was no significant difference in any of the criteria examined.—*J. Dai. Sci., May, 1953.*

Experimental Evaluation of Culture and Serum Vaccination for the Control of Swine Erysipelas

IV. Gilts Vaccinated with Culture and Serum Before Breeding, and Its Immunizing Effect on Their Offspring

RICHARD D. SHUMAN, B.S., D.V.M.

Washington, D.C.

SINCE BABY PIG vaccination in the swine section herd, U.S.D.A. Agricultural Research Center, Beltsville, Md., was shown to be incapable of protecting pigs up to market age,[1] and since treatment with culture and serum as baby pigs and again as weanlings would not be economical, the problem of protecting pigs after birth up to vaccination as weanlings still remained. For this reason, the possibility of transference of immunity from dam to offspring was considered, and if it did occur, approximately how long would it persist in the baby pigs.

Peterman *et al.*[2] did recommend, as part of their control program, a procedure of vaccinating the gilts soon after they have been selected for breeding purposes. Van Es and McGrath[3] mention an early observation by Frohner and Zwick, "that sucking pigs nursed by brood sows involved in erysipelas, as a rule, escape infection even if the causative microbes are eliminated with the milk." This was interpreted as a transference of immunity from sow to litter, and quite accidentally we were able to make a very similar observation. A sow tested by skin scarification was found to be susceptible. Unknown to us, this scarification procedure occurred during her gestation period, as thirty-six days later she farrowed 9 normal pigs and 1 dead pig. One of these pigs was scarified five days after birth for the purpose of establishing the status of the litter to swine erysipelas infection, while the balance of the litter was vaccinated with culture and serum. The scarified pig was found to be completely immune to percutaneous infection.

MATERIALS AND METHODS

For this experiment, young gilts were obtained from the swine brucellosis project. They had been raised on the premises of the Animal Disease Station, Beltsville, Md., from boars and sows which originated from the swine section herd. The young gilts represented the second farrowing of these sows. These gilts remained in what is named the "clean area" of the Station until maturity, and were then placed in an experimental barn for vaccination and subsequent breeding. In previous tests, pigs from the "clean area" had demonstrated susceptibility to both hog cholera and swine erysipelas.

The time of vaccination of the gilts was carried out with the idea of their maturity and not with particular reference to the time of breeding, except that vaccination was accomplished before breeding. Eight gilts were used in this experiment, 6 being vaccinated and 2 left unvaccinated to serve as controls. The regular label dosage for hogs over 100 lb. was used, e.g., 1 cc. of culture with 20 cc. of serum. Company A swine erysipelas antiserum, serial lot 3, and company C desiccated live culture, serial lot 2, were used; both products having passed the Bureau of Animal Industry standard tests "for release." All animals were on a basic ration containing an antibiotic (aurafac) at the rate of 6 lb. per ton of feed. As the baby pigs were on concrete floors, they were given reduced iron tablets shortly after birth.

The gilts were placed together in one large pen in the experimental barn. For a while the boar was introduced daily, later on continuously, until no further interest was manifested. Prior to farrowing, each gilt was placed in an individual pen, and the only other pigs that went into this barn were culture control pigs for the various exposures. These culture control pigs also originated from the "clean area" of the Animal Disease Station.

Demonstration of either immunity or susceptibility was again based on the percutaneous or skin scarification method of exposure.[4] The four lyophilized strains, S192, HC585, 422-1, and NF4, used and the interpretation of the result of scarification were the same as outlined in the preceding baby and weanling pig vaccination work (*see* October JOURNAL, pp. 304-310).

The skin scarification exposure studies of these vaccinated gilts and their litters for either susceptibility or immunity included two aspects: (1) the exposure of the nursing litters from three to fifty-eight days after farrowing, and (2) the

From the Pathological Division, Bureau of Animal Industry, Washington, D. C.

exposure of the gilts, which was carried out at the same time as their respective litters.

EXPERIMENTAL RESULTS

Complete immunity of the litters from the vaccinated gilts prevailed up to and including the sixth week (45 days) after farrowing. However, the litter scarified at eight weeks (58 days) had only 4 of 11 pigs showing complete immunity (I), 1 pig having a borderline reaction called an incomplete immunity (II), and the remaining 6 pigs showing susceptibility with only local reactions (table 1). Complete immunity was found in the 6 vaccinated gilts when they were tested between the fourth and eighth month after vaccination (table 2). Both nonvaccinated controls, 9715 and 9736, showed susceptibility followed by generalization of the infection. The litter from 9715, when exposed along with the dam eleven days after birth, showed complete susceptibility followed by the death of 5 of 7 baby pigs. *Erysipelothrix rhusiopathiae* was recovered from all 5. The 2 surviving pigs never fully recovered from the effects of scarification and remained small and rough looking. All pigs in the litter of 9736 were susceptible, with 6 showing local reactions and 1 showing generalization when scarified forty-five days after birth. All 6 animals included in this study as culture controls were susceptible to the scarification procedures, 2 showing only local reactions, and 4 a generalization of the infection (table 2).

DISCUSSION

The duration of immunity found to be prevailing in the litters from the vaccinated gilts came as a surprise, especially when the lapse of time from vaccination of the gilts to exposure of the litters was considered. Not only was a very substantial duration of immunity produced in the gilts, but there was also a persistence of immunity in their offspring that extended almost to weaning time.

A comparison of the results following scarification of animals vaccinated as baby pigs or as gilts illustrated the fact that age at the time of vaccination must play an important role in the animal's response to immunization procedures, the best response being seen in the older vaccinated animals. It would also be worth-while to determine the duration of immunity in animals vaccinated at maturity.

TABLE I—Scarification of Baby Pigs Farrowed by Gilts Vaccinated at Maturity

Gilt (No.)	Vaccination date	Farrowing date	Baby pig (No.)	Scarification date	Age of pigs at time of scarification	Exposure cultures				Final status of baby pigs	Remarks
						S192	HC585	422-1	NF4		
9742	2-21-52	6-14-52	1 to 5	6-17-52	3 days	I	I	I	I	I	All immune
9739	2-21-52	6-14-52	1 to 7	7-8-52	24 days	I	I	I	I	I	All immune
9737	2-21-52	7-2-52	1 to 6	7-30-52	28 days	I	I	I	I	I	All immune
9741	2-21-52	7-5-52	1 to 7	8-19-52	45 days	I	I	I	I	I	All immune
9714	2-21-52	8-13-52	1	10-8-52	58 days	S	S	S	S	S(L)	None
		8-13-52	2	10-8-52	58 days	I	I	I	I	I	None
		8-13-52	3	10-8-52	58 days	S	S	S	S	S(L)	None
		8-13-52	4	10-8-52	58 days	S	S	S	S	S(L)	None
		8-13-52	5	10-8-52	58 days	I	I	I	I	I	None
		8-13-52	6	10-8-52	58 days	S	S	S	S	S(L)	None
		8-13-52	7	10-8-52	58 days	I	I	I	I	I	None
		8-13-52	8	10-8-52	58 days	S	S	S	S	S(L)	None
		8-13-52	9	10-8-52	58 days	S	S	S	S	S(L)	None
		8-13-52	10	10-8-52	58 days	I	I	I	II	II	None
		8-13-52	11	10-8-52	58 days	I	I	I	I	I	None
9715	Not Vac.	6-27-52	1	7-8-52	11 days	S	S	S	S	S(G)	Stunted
		6-27-52	2	7-8-52	11 days	S	S	S	S	S(G)	Stunted
		6-27-52	3	7-8-52	11 days	S	S	S	S	S(G)	Died
		6-27-52	4	7-8-52	11 days	S	S	S	S	S(L)	Died
		6-27-52	5	7-8-52	11 days	S	S	S	S	S(L)	Died
		6-27-52	6	7-8-52	11 days	S	S	S	S	S(L)	Died
		6-27-52	7	7-8-52	11 days	S	S	S	S	S(L)	Died
9736	Not Vac.	7-5-52	1	8-19-52	45 days	S	S	S	S	S(L)	None
		7-5-52	2	8-19-52	45 days	S	S	S	S	S(L)	None
		7-5-52	3	8-19-52	45 days	S	S	S	I	S(L)	None
		7-5-52	4	8-19-52	45 days	S	S	S	II	S(L)	None
		7-5-52	5	8-19-52	45 days	S	S	S	S	S(G)	None
		7-5-52	6	8-19-52	45 days	S	S	S	I	S(L)	None
		7-5-52	7	8-19-52	45 days	S	S	II	II	S(L)	None

I = Complete immune; II = incomplete immune; S = susceptible; S(L) = susceptible local; S(G) = susceptible generalized.

Scarification of the litters from the 2 nonvaccinated gilts suggests that age is a factor in the susceptibility of pigs to percutaneous infection. As can be seen in table 1, all the pigs of gilt 9715, when tested at 11 days of age, were not only susceptible to the four exposure strains, but 5 out of 7 pigs died, with the other 2 showing generalization of the infections. The litter of 9736, though showing susceptibility at 45 days of age, presented an individual variability of the cutaneous reactions to the four exposure strains; as well as only 1 pig showing generalization, the remaining 6 reactions were localized. Had this been a vaccinated litter, it could easily be interpreted as demonstrating a small degree of immunity, e.g., a "petering out" of immunity on a litter basis. These 2 litters do not provide sufficient evidence to form a definite conclusion, but they do indicate that age may be a factor associated with susceptibility.

Antibiotic feeding at this level certainly did not appear to influence the result of scarification as judged by the susceptibility of the nonvaccinates and their litters as well as the culture controls in this experiment.

The demonstration of immunity to percutaneous infection of pigs from vaccinated gilts, both in the 5-day-old pig and in the litters reported, presents a matter for very interesting speculation. In other words, just what is the mechanism by which immunity was developed in these litters.

All the pigs of these litters were subsequently killed and examined for atrophic rhinitis, and all were found to be negative.

TABLE 2—Scarification of Gilts Vaccinated at Maturity

Gilt (No.)	Vaccination date	Age time of vaccination	Approx. breeding date	Farrowing date	Scarification date	Scarified after vac. in days	Exposure Culture				Final status
							S192	HC585	422-1	NF-4	
9742	2-21-52	8 months	2-21-52	6-14-52	6-17-52	117	I	I	I	I	I
9739	2-21-52	8 months	2-21-52	6-14-52	7-8-52	138	I	I	I	I	I
9737	2-21-52	8 months	3-11-52	7-2-52	7-30-52	160	I	I	I	I	I
9741	2-21-52	9 months	3-14-52	7-5-52	8-19-52	180	I	I	I	I	I
9740	2-21-52	8 months	Barren	—	9-19-52	211	I	I	I	I	I
9714	2-21-52	9½ months	4-22-52	8-13-52	10-8-52	230	I	I	I	I	I
9715	Not vac.	—	3-5-52	6-27-52	7-8-52	—	S	S	S	S	S(G)
9736	Not vac.	—	3-14-52	7-5-52	8-19-52	—	S	S	S	S	S(G)
Culture Controls for Gilts and Litters											
120					6-17-52	—	S	S	S	S	S(L)
123					7-8-52	—	S	S	S	S	S(L)
7943					7-30-52	—	S	S	S	S	S(G)
9913					8-19-52	—	S	S	S	S	S(G)
144					9-19-52	—	S	S	S	S	S(G)
148					10-8-52	—	S	S	S	S	S(G)

I = Complete immune; II = incomplete immune; S = susceptible; S(L) = susceptible local; S(G) = susceptible generalized.

Antibiotic feeding at this level certainly did not appear to influence the result of scarification as judged by the susceptibility of the nonvaccinates and their litters as well as the culture controls in this experiment.

The demonstration of immunity to percutaneous infection of pigs from vaccinated gilts, both in the 5-day-old pig and in the litters reported, presents a matter for very interesting speculation. In other words, just what is the mechanism by which immunity was developed in these litters.

All the pigs of these litters were subsequently killed and examined for atrophic rhinitis, and all were found to be negative.

SUMMARY

Baby pigs from gilts that had been vaccinated with culture and serum when mature, but before being bred, were immune to percutaneous infection up to and including the sixth week (45 days). Some protection was afforded up to the eighth week (58 days). Although based on a limited number of experimental animals, this suggests a method for protecting pigs against swine erysipelas from birth to the weaning period. Vaccination of mature gilts with culture and serum resulted in an immunity to percutaneous infection from 4 months of age (117 days) to at least 8 months (230 days).

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Penicillin seemed of little value for pneumonia secondary to canine distemper but streptomycin does seem to have value.—*H. C. Stephenson, D.V.M., Ithaca, N. Y.*

Treatment of Infectious Ovine Keratitis

WILLIAM F. RILEY, Jr., D.V.M., and RALPH D. BARNER, D.V.M.

East Lansing, Michigan

IN RECENT publications,[1,2] the existence of a pink-eye type of ophthalmia in Michigan sheep herds has been reported. The fact that this particular condition has not been common in sheep in this state was borne out by a search through existing records and by talking to many practitioners throughout the state. Herdsmen and veterinarians concerned with the recent outbreaks have reported that never before to their knowledge had sheep in their localities suffered from a similar condition.

The first recognizable incident[3] of a clinically parallel entity in this area occurred during the winter months of 1949. At that time, a flock of approximately 50 sheep was showing symptoms of keratitis. The owner had been trying various drugstore remedies for about two weeks before consulting our ambulatory service. Examination of this flock disclosed some 18 to 20 head visibly affected with varying degrees of conjunctivitis and keratitis. Several were completely blind, having bilateral opacities of the cornea. It was because of these latter cases that the owner became alarmed enough to ask for assistance. A herd of 16 grade dairy cows on the same farm failed to show any symptoms throughout this outbreak.

Treatment was instituted along the usual lines as for infectious bovine keratitis. Results were slow. The greater portion of two months passed before the symptoms abated, and a wide variety of antibiotics and antiseptic agents were instilled into the eyes of these sheep. No attempt was made at that time to determine the presence of any specific causative agent.

When similar outbreaks began to appear in scattered localities in Michigan during the early winter of 1950 and the winter and spring of 1951,[1] our interest was aroused and a search was instituted to determine the causative agent. Examination of the flocks involved failed to disclose any obvious departures from good feeding and management practices. Careful examination failed to disclose the presence of ectoparasites. The season obviated fly transmission. Accordingly, prompted by the findings of Johnson,[4] Boughton and Hardy[5] in the United States, Blakemore[6] in England, and various other workers cited in our initial report,[1] steps were taken to identify the causative agent in our Michigan outbreaks. More recently, Newsom,[7] in his text has included infectious keratitis of sheep as a specific contagious disease.

The findings as reported in the four farm flocks initially studied have been duplicated in new outbreaks in other flocks within the past winter months (1952-1953).[2] Scrapings of the conjunctivae in these new outbreaks showed the epithelial cells of some to contain inclusion bodies comparable to those seen in last year's outbreak. While studies to date have failed to accomplish artificial cultivation of *Rickettsia conjunctivae* (later renamed *Colesiota conjunctivae* by Rake[8]), still its presence, both in the United States[1, 4, 5] and abroad[6, 9, 10] in cattle and sheep affected with keratitis, leads one to suspect it as the true etiological factor.

Further supporting our suspicions of this particular organism is the fact that clinically it was noticeable that the usual medicaments successfully employed to overcome infectious bovine keratitis, caused by a bacterial agent, *Moraxella bovis*, proved very much less efficient in clearing up the primary keratitis.

Symptoms.—Lacrimation was the first sign the herdsman noticed in new cases. It was usually profuse and bilateral. The conjunctiva was found to be hyperemic and sensitive. Photophobia was noticeable in the early stages of the disease. Peripheral vascularization (pannus) of the superficial corneal layers followed and in a few days complete bilateral opacity was established. The entire cornea assumed a bluish white color. The animal was quite blind when the disease progressed to this extent. Ulceration of the cornea was present in a few of the advanced cases. It is not known whether this latter phenomenon is a result of the inflammatory process or attributable to injury as a result of being blind. At no time during the course of this disease has a purulent exudate been noted.

From the Department of Surgery and Medicine (Riley) and the Department of Animal Pathology (Barner), School of Veterinary Medicine, Michigan State College, East Lansing.

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Appreciation is hereby acknowledged to Parke, Davis & Company for supplying the chloromycetin ointment necessary for therapy.

Course.—Some attacks have been relatively mild, running a five- to seven-day course with a morbidity of approximately 10 to 15 per cent. However, the morbidity has usually averaged 20 to 50 per cent in untreated flocks. The infection has persisted for thirty to sixty days. Improvement under general treatment is the rule, but the keratitis and conjunctivitis usually persists much longer than one would expect. Segregation, regardless of treatment, is instrumental in reducing the morbidity. Direct contact and droplet infection seem to be the mediums by which the disease is spread.

Treatment.—Early therapy consisted of segregation, protection from wind and sunlight, and the application of various antiseptic and antibiotic preparations. Briefly, some of the latter were metaphen (1:3,000) ointment, sulfathiazole (5%) ointment, atropine (1%) ointment, argyrol (10%) ointment, penicillin G potassium (30,000 I.U. per gram) ointment, and penicillin G and dihydrostreptomycin (20,000 I. U. penicillin and 13 mg. dihydrostreptomycin per gram) ointment. Astringent collyriums (berberine sulfate, boric acid, and zinc sulfate) as well as various powders containing sulfathiazole, sulfanilamide (5 to 10 % of each), and boric acid (20 to 23 %) were given a fair trial. Injections of sterile milk or mixed bacterins (*Pasteurella boviseptica*, Streptococcus, Corynebacterium and Staphylococcus) gave indifferent results.

As a result of the studies on the etiology in these particular outbreaks, it was decided to use chloromycetin® in some form for therapy. Tatum[11] reported on the effective use of chloromycetin in rickettsial diseases in 1948. Accordingly, chloromycetin (chloramphenicol, Parke-Davis, a crystalline antibiotic identified chemically as D-(-)-threo-2-dichloroacetamido-1-*p*-nitrophenyl-1, 3-propanediol) was procured in an ointment base suitable for instillation into the eye. Specifically, ointment chloromycetin, veterinary type "A", 200 mg., was used. Locally, this particular ointment quickly softened and spread, forming a thin film to adequately contact all portions of the conjunctiva and cornea. On the basis of the limited trials herein reported, it was felt that the type "A" base was satisfactory as an ophthalmic ointment base. Various laboratory trials[12] have conclusively proved that this particular ointment base releases its antibiotic content with extreme rapidity. This is a most important factor in clinical medicine.

Ointment chloromycetin, veterinary type "A", was applied twice daily in severely affected cases, and once daily in mild cases. As a rule, mild cases showed improvement within two to three days, and appeared normal in about one week. More severe cases required applications twice daily for four or five days, after which once daily for another four or five days proved sufficient in most animals. The degree of clinical improvement served as a guide to the frequency and the duration of treatment. Segregation was practiced in every possible instance. In those flocks where the outbreak was immediately preceded by the purchase of purebred breeding stock (often a new ram) from some outside source, it was felt desirable to medicate those newly purchased animals also, even though they were not visibly affected. It was felt that in this way the danger of the carrier state[6] might be minimized.

SUMMARY AND CONCLUSIONS

1) A limited clinical evaluation of the effectiveness of various therapeutic agents commonly employed in inflammatory diseases of the eye was carried out in cases of infectious ovine keratitis.

2) Chloromycetin* was used, in an ointment base, having a concentration of 200 mg. of chloromycetin in 3.8 Gm. of ointment.

3) Preliminary results indicate chloromycetin to be effective in the treatment of infectious ovine keratitis.

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*Since this article was submitted, several practicing veterinarians in Michigan have reported chloromycetin ointment to be most effective treatment in cases of pinkeye in cattle which did not respond to other therapeutic agents.

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Heat Tolerance of Red Sindhi Cattle

To test their comparative tolerance to high temperatures and humidities, 15 thoroughbred Holstein-Friesians and 15 crossbred (Red Sindhi-Holstein-Friesian) nonlacting heifers were exposed to a series of elevated temperatures and humidities in a climate control chamber. Five of each breed were enclosed for eight hours or until their temperature reached 107 F. Their temperature, respiration, and pulse rate were recorded hourly.

At 75 F., there was no difference in the temperature of the breeds but the Holstein-Friesians had higher respiration and lower pulse rates. At all elevated temperatures and humidities, crossbreds exhibited significantly lower temperature and respiration rates but higher pulse rates. All Holstein-Friesians were removed with temperatures of 107 F. or above during one or more trials but no crossbred ever attained a temperature of 105 F. The response of both breeds was greater to humidity than to temperature. —*J. Dai. Sci., June, 1953.*

The Ubiquity of Toxoplasmosis

The range of animal species that can be infected with Toxoplasma is unique for the Protozoa, all mammals and birds investigated having been found susceptible. It is probably the most ubiquitous and prevalent of parasites. The most commonly recognized human toxoplasmosis manifestations are the result of congenital infections. They are chorioretinopathy, cerebral calcification, hydrocephalus or microcephalus, and psychomotor disturbances.

It is believed that only mothers with primary acute infection can transmit Toxoplasma organisms to their offspring *in utero*. In one study, 50 mothers who had given birth to one child with congenital toxoplasmosis subsequently gave birth to one or more normal children. Following uterine transmission, the fetus may die *in utero* or after an abortion, or it may be born prematurely or at term with active toxoplasmosis, or it may be normal at birth and develop symptoms days to months afterward. The sulfonamides, especially sulfadiazine, show activity against toxoplasmosis in mice and rabbits. Antibiotics even at extremely high dosages have shown only slight effect. Recent studies suggest that Toxoplasma organisms are often harbored but rarely cause serious damage, an instance of a lot of infection but little disease.—*J.Am.M.A., Aug. 22, 1953.*

Toxoplasmosis in Mental Deficiency.—When 700 mentally deficient patients were tested, about 8 per cent gave toxoplasmin skin and dye reactions. The incidence increased with age. Because it was infrequently associated with clinical toxoplasmosis, the latter is not considered a common cause of mental defects in man.—*J. Am. M. A., July 25, 1953.*

Chlortetracycline for Toxoplasmosis

When healthy persons in Germany were examined with the Sabin-Feldman test for toxoplasmosis, 2 per cent reacted; of those suspected of having the disease, 18.6 per cent reacted. Ten of 13 stillborn infants were positive as were 3 of 8 fetuses from miscarriages. The titers of 2 cases of laboratory infection became negative after two or three weeks when treated with chlortetracycline (aureomycin).—*Vet. Bull., July, 1953.*

Penicillin for Fowl Cholera.—Pasteurella multocida affected 700 fowl in two outbreaks and 27 geese in the third outbreak. They were successfully treated by intramuscular injections of penicillin. Two of these outbreaks were traced to wild geese. —*Vet. Bull., July, 1953.*

Hypertrophic Pulmonary Osteoarthropathy Resulting from Metastasis to the Lungs in Dogs

ROBERT L. LEIGHTON, V.M.D., and JOSEPH M. STOYAK, V.M.D.

Springfield, Massachusetts

IN THE last decade, a number of reports of the occurrence of hypertrophic pulmonary osteoarthropathy have appeared in the literature.[1-11] Several authors have ably discussed the histology and pathology of this condition and their articles are highly recommended.

It is the purpose of this paper to report on a case of the disease which resulted from tumor metastasis from the mammary gland to the lung, and to bring attention to other cases reported in the literature in which metastasis to the lungs was involved.[1,2] In this, we must take exception to the following statement in point 5 in the discussion in the recent article by Mather and Low:[9] *Chronic pulmonary osteoarthropathy does not appear to be associated with metastatic neoplastic involvement of the lung. . .*" (Italics ours.)

In a study of 4 cases at the Angell Memorial Animal Hospital in 1950,[2] H. Stunzi reported 2 of them to be associated with lung metastases, 1 from a fibrosarcoma of the pleura, the other from an adenocarcinoma of the perianal gland. Schnelle[1] reports a case which was metastatic from a tumor of the prostate.

The following report is given as an addition to the literature and as a case in point.

CASE REPORT

On Sept. 24, 1952, an 11½-year-old female Springer Spaniel was brought into our clinic for euthanasia. The dog was *in extremis* and died immediately. Examination of the body revealed a markedly advanced case of hypertrophic pulmonary osteoarthropathy. Because of delay in obtaining the body for autopsy, it unfortunately was not possible to perform any laboratory examination of blood or urine.

In March, 1951, the dog had had a breast tumor removed. This recurred and a 3-lb. mass was removed one year later after which the owners observed a gradual loss of weight, decreased activity, depression, and lameness. No particular diminution in appetite was observed, nor did the dog evince any cough or marked dyspnea. The time of actual onset of the thickening of the extremities could not be stated, although the actual period of extreme enlargement must have occurred in the last few months.

Autopsy Report.—The dog had been in only fair condition. Her matted, filthy hair, the long nails, and the multitudes of fleas gave evidence of prolonged neglect. Closer examination of the limbs revealed a marked thickening extending from below the elbow and stifle to, and involving, the distal extremities. Just below the tarsal and carpal joints, the legs were more than 9 cm. in diameter.

Posterior to the right third mammary gland was a healed suture line where the posterior mammae of that side had been removed. A growth about 7 cm. in diameter was present in the position of the third mammary gland. No other external abnormalities were seen.

There was a small amount of subcutaneous fat. When the skin was removed, the mammary tumor was found to be covered with a glistening capsule. Cut sections revealed a tough, firm, yellowish red vascular tissue.

The gall bladder was thin-walled, enormously distended, 10 cm. by 20 cm., dark red, and contained about 600 cc. of blackish bile. The liver was swollen and dark mahogany in color. The spleen contained a nodule 3 cm. in diameter. The pancreas contained a mass 1½ cm. in diameter. The ovaries were a mass of glistening, transparent cysts.

The bladder wall was markedly thickened, with a highly vascular surface. The stomach and intestines were normal. The kidneys displayed a number of rounded enlargements under the capsule. On cut section, these were revealed to be round tumor masses up to 2½ cm. in diameter. The capsule peeled with some difficulty. The

From the Rowley Memorial Hospital, Springfield, Mass.

Fig. 1—Anteroposterior and lateral radiograph of forelimbs showing appearance of bone growth in a case of hypertrophic pulmonary osteoarthropathy.

Fig. 2—Anteroposterior and lateral view of the hind limb.

tumor masses were firm, yellowish red and were located in the renal cortex.

When opened, the thoracic cavity revealed a pleura studded with many small

Diagnosis.—Mixed tumor, malignant, right third mammary gland, with metastasis to lung and kidney, canine.

Secondary hypertrophic pulmonary osteoarthropathy. Follicular cyst, multiple ovary.

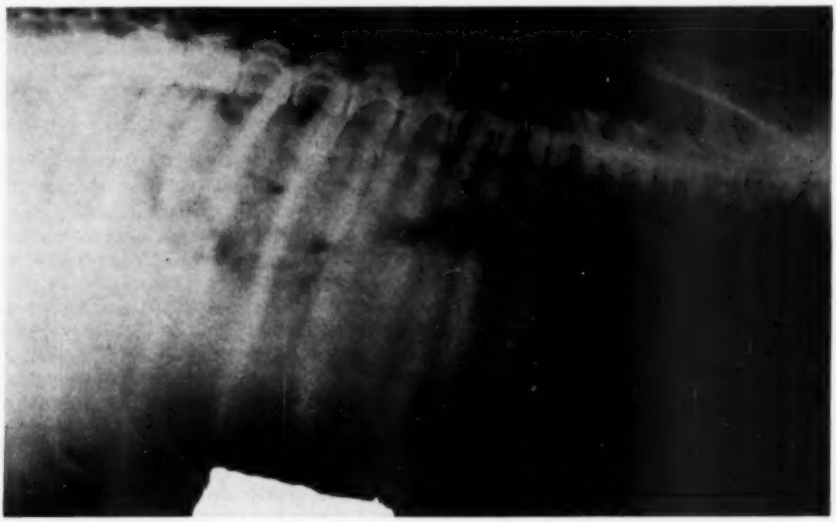

Fig. 3—Lateral view of chest showing lung area replaced with dense tumor tissue.

nodules, and almost all of the lung tissue was replaced by lobulated masses of tumor tissue. There was so little normal lung tissue left that it seemed incredible that the animal could have lived as long as it did. The bronchi and trachea were filled with frothy foam. The heart was enlarged and flabby.

Examination of the limbs revealed a tremendous bilateral thickening of the extremities, a doubling of size below the hock and carpus. Motion of the joints involved was considerably limited. There was a markedly thickened joint capsule in the hock, with an increase in joint fluid. The subperiosteal bone growth was firm and seemed to envelope the normal structures. All limbs were similarly affected. Roentgenograms were taken of the front and hind limbs and of the chest (fig. 1, 2, 3).

Histological Reports.—Sections of various affected tissues were sent to the Armed Forces Institute of Pathology and to the School of Veterinary Medicine, University of Pennsylvania.

We quote from a portion of the A.F.I.P. report:

Likewise, the pathology department of the University of Pennsylvania School of Veterinary Medicine found the tissue to be primary in the mammary gland with metastasis to the lungs. They agree that this is a definite case of pulmonary osteoarthropathy.

SUMMARY

A case of hypertrophic pulmonary osteoarthropathy is reported in which the lung tumor was metastatic from a mammary tumor. Similar cases are cited in the literature. It, therefore, seems clear that hypertrophic pulmonary osteoarthropathy can be associated with metastatic neoplastic involvement of the lung.

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A Study of Hardware Disease

The *Livestock Conservation News* (Sept., 1953) announces that a committee is making a study of hardware disease (traumatic gastritis). The Meat Inspection Service, U. S. D.A., reports 31,604 cattle condemned for this disease in four years, an annual loss of well over $1,000,000. This does not include animals which die or are butchered elsewhere. Preliminary reports indicate a variable distribution of cases, 60 per cent of the total being in 17 per cent of the plants reporting.

Johne's Vaccine and Tuberculosis

Varying degrees of cross protection occur between different pathogenic acid-fast organisms. When Johne's vaccine was used on 14 of 28 young, healthy goats, all developed vaccinal nodules. The other 14 were controls. Three months later, all except 2 controls were given 5 drops of bovine tubercle bacilli suspension in both conjunctival sacs. When some of each group were autopsied five, eight, and eleven, and twenty months, respectively, after exposure, all were infected but the vaccinated much less so than the controls. One control died of generalized tuberculosis four months and another eight months after exposure.

When autopsied, only 1 control failed to show thoracic involvement, while by the eighth month, only 1 of 11 vaccinated goats showed involvement beyond the nodes of the head. However, by the twentieth month, 2 of the 3 vaccinated goats showed thoracic involvement. Vaccination apparently only retarded the spread of the infection. When 13 of the vaccinated and artificially inoculated goats were intradermally tested on opposite sides of the neck with mammalian and avian tuberculin, there was little difference in the reactions.—*Brit. Vet. J., July, 1953.*

Progress with Bluetongue in Sheep

The Sonora substation of the Texas Agricultural Experiment Station reports progress in its investigations of bluetongue virus disease of sheep. An epizoötic swept the sheep-raising area of Texas in the summers of 1948 and 1951. The mortality was less than 1 per cent but screw-worm infestation, fleece impairment, and other factors made the total loss significant. The disease appeared on only a few ranches in 1952 but has been widespread during the summer of 1953. Since a night-flying insect, Culicoides spp., is a vector of this virus in South Africa, the U. S. Bureau of Animal Industry has undertaken a taxonomic survey of this genus in the Southwest.

Recently at Sonora, clinical bluetongue was produced by the injection of Culicoides which had been caught in the area. Because there seems to be more than one strain of the virus, infective material has been sent to California, where the disease occurs in a more severe form, for cross-immunity tests. Results in attenuating the virus by chicken embryo passage are encouraging.—*D. A. Price, D.V.M., and W. T. Hardy, D.V.M., Sonora, Texas.*

Sensitization to Antibiotic

Early in 1953 many nurses, who were giving injections of streptomycin, were reported to be sensitized to this drug. Therefore, a drug-sensitization survey was made in 146 hospitals and health stations. Of these, 86 reported cases with skin manifestations such as urticaria, eczema, dermatitis, erythema, etc. In almost every case, the hands, arms, face or eyes were affected. Of the cases reported, 188 were sensitized to streptomycin, 80 to penicillin, and 1 to chloramphenicol.—*Brit. M. J., July 4, 1953.*

Mucormycosis in Animals

A Report of Three Cases

LIEUTENANT COLONEL CHESTER A. GLEISER, V.C., U.S. Army

Washington, D. C.

THE PHYLUM, Thallophyta, within the plant kingdom is made up of two large divisions —the algae which have chlorophyll, and the fungi which are without chlorophyll. The fungi are broken down into the Pseudomycetes, or false fungi, and the Eumycetes, or true fungi. The Phycomycetes, a class of true fungi, include the order, Mucorales which, in turn, contains the family, Mucoraceae. Within this family at least one species, *Mucor corymbifer*, is occasionally parasitic in man and animals.[2,4] The Mucorales are characterized by nonseptate hyphae and by the development of asexual sporangiospores, by progressive cleavage within sporangia, and by zygospores.[4] The continuous, coarse, branching, nonseptate mycelium is the one most distinguishing feature of Mucor in animal tissues.

Lichtheim,[11] in 1884, reported the isolation of *Mucor rhizopodiformis* and *Mucor corymbifer* from moistened bread, and by intravenous injection of large numbers of these spores into rabbits established their pathogenicity. Lesions were found in the kidneys, lymphoid tissues of the intestine, and the mesenteric nodes. His observations are justifiably open to question in view of the artificial method of exposing these animals.

Paltauf,[14] in 1885, reported a case of human mucormycosis with focal abscesses in the lungs, cerebrum, cerebellum, pharynx, and ileum. Ulcerated nodules were present in the intestine.

Lindt,[12] in 1886, isolated two other species of Mucor from moistened bread, *Mucor pusillus* and *Mucor ramosus*. He demonstrated the pathogenicity of the spores of these species by the same techniques that Lichtheim had used.

Theobald Smith,[17] Bendixen and Plum,[1] Gilman and Birch,[8] and Jungherr,[10] all reported isolation of Mucor organisms from fetal tissues or placenta in bovine abortions not caused by *Brucella abortus*. Smith[17] produced bilateral kidney lesions in rabbits by the intravenous injection of the spores of the Mucor he had isolated, and Gilman and Birch[8] produced placental infection with and without abortion by inoculating 5 pregnant cows with the Mucor they had obtained. They described the principal lesion of the placenta in their spontaneous cases as necrosis of the cotyledons with hemorrhage. Jungherr[10] also described the necrotic lesions of the cotyledons in his cases.

Christiansen[3] and Nielson[13] observed 9 cases of mucormycosis in swine and described intestinal and mesenteric lesions. There is some doubt as to the authenticity of this report because, as Saunders[16] pointed out in his review, the authors stated that examination of wet preparations of infected tissue revealed clearly defined septums in the mycelium. Emmons,[6] however, expressed the opinion that Mucor may become septate under certain conditions and has demonstrated septate hyphae to the author. Saunders[16] also mentions the isolation of *M. corymbifer* from the abscesses of an alpaca and the submaxillary nodes of a pig.

Gregory, Golden, and Haymaker,[9] in 1943, reviewed the literature on mucormycosis, and reported 3 cases of human mucormycosis of the central nervous system. They noted that in the majority of reported human infections, only a single organ or system is involved, most of the infections being in the lungs or ears, although in 5 cases gastric ulcers were thought to be due to Mucor. The central nervous system appears to be an infrequent site of infection.

Because of the lack of unfixed tissues from the infected areas of brain and meninges, cultures of the fungus could not be obtained in any of the cases studied by these authors. The tissue sections, however, were reviewed by Dr. C. W. Emmons,* who expressed the opinion that the organisms were morphologically compatible with Mucor. Two of the patients were known to have diabetes and the third was believed to be a diabetic on the basis of the clinical history available and histological examination of tissue. There was no other common factor in these patients. Lesions of mucormycosis were present in the eyes of 2 of these patients and it was proposed that in all 3 cases the fungi had gained entry by way of the orbit. The authors noted the tendency of the fungi to invade the walls of blood vessels and to grow within their lumina.

Friedenwald, Wilder, *et al.*,[7] demonstrated the presence of the typical hyphae of Mucor in the choroid and ciliary body of human eye with a mild lymphocytic reaction (AFIP Acc. 87679).

Conant[4] stated that the genus Mucor is a common laboratory contaminant, and that it may be a secondary invader, but only rarely a primary invader. He advocated that the diagnosis of mucormycosis be made "reluctantly and only when the evidence is overwhelming." Dodge[5] expressed the same view in 1935 when he stated that there

From the Armed Forces Institute of Pathology, Washington, D. C.

The author is grateful to Dr. C. W. Emmons, of the National Institutes of Health, for reviewing these cases and this manuscript.

*C. W. Emmons, Ph.D., principal mycologist, National Microbiological Institute, National Institutes of Health, Bethesda, Md.

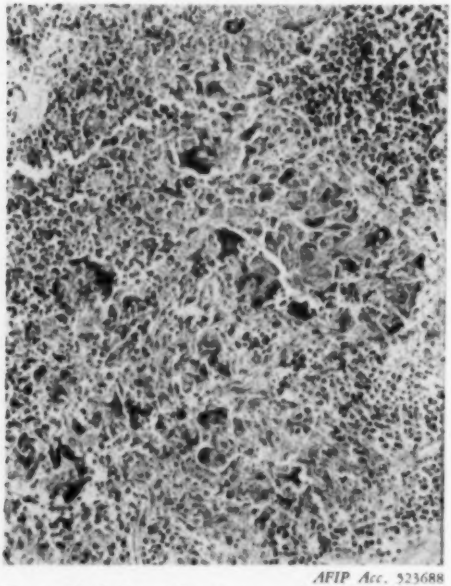
was little conclusive evidence that this typically saprophytic genus (Mucor) was pathogenic for man. He classified *M. corymbifer* as *Absidia corymbifer* and discussed it separately.

CASE REPORTS

The purpose of this paper is to report three cases of mucormycosis in animals. Fresh material for cultural examination was not available, but these cases, like those of Gregory and associates, were reviewed by Dr. C. W. Emmons, who expressed the opinion that the fungi in each case were morphologically compatible with Mucor.

*Case 1 (AFIP Accession 523688).***—A 3½-year-old spayed English Setter was dying when brought to the clinic. Emaciation was advanced, and the rectal temperature was 98.4 F. There was no response to supportive treatment and terramycin.® Several convulsions occurred on the second day after admission and the dog died.

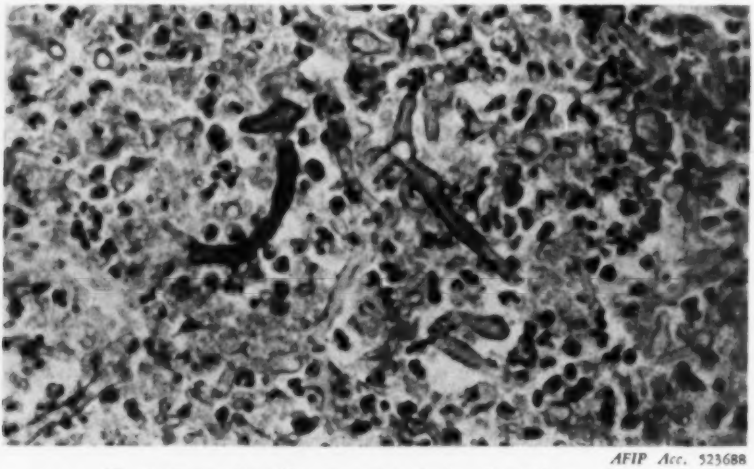
At autopsy, the left kidney was found to be greatly altered and enlarged. The capsule stripped with difficulty; the cortex and medulla appeared to be "degenerated"; the pelvis was completely "broken down." The right kidney was grossly normal. No other changes were observed. Microscopically, there was thickening and infiltration of the renal capsule with lymphocytes, plasma cells, and an occasional giant cell. The entire renal architecture was disrupted by the extensive granulomatous and necrotic processes within the cortex, medulla, and pelvis. Numerous large masses of plasma cells and lymphocytes were seen in the cortex and in the medulla. In addition, the numerous granulomatous foci were characterized by necrosis and the presence of many multinucleated giant cells. Within these foci, bizarre nonseptate hyphae were abundant



AFIP Acc. 523688

Fig. 1—Marked granulomatous reaction with numerous multinucleated giant cells in kidney of a dog. x 128.

**Contributed by Dr. R. L. Leighton, Rowley Memorial Hospital, Springfield, Mass.



AFIP Acc. 523688

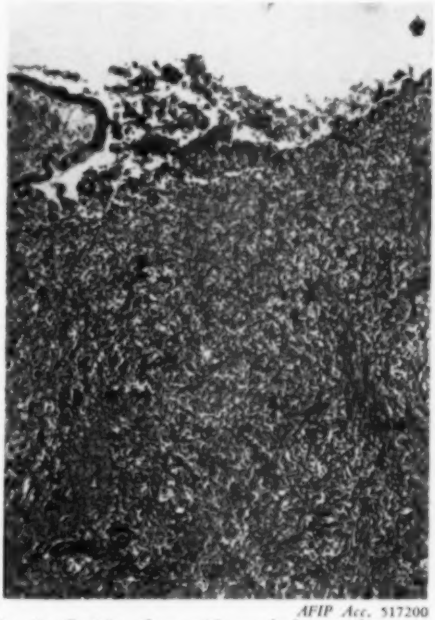
Fig. 2—Coarse branching nonseptate hyphae in a kidney. x 570.

and were frequently observed within the giant cells (fig. 1, 2). The process was much more extensive in the medulla than in the cortex, and still more prominent in the pelvis where there was marked neutrophilic infiltration into the large area of necrosis in which similar, branching, nonseptate hyphae were observed. Much of the renal parenchyma had been totally obliterated by this extensive process.

The secondary changes in the glomeruli and tubules consisted chiefly of thickening of the Bowman's capsules and basement membrane. Vascular invasion as described by Gregory, Golden, and Haymaker[9] was not seen. The opposite kidney had several interstitial foci of lymphocytes and plasma cells. Some of the Bowman's capsules were thickened and there were numerous proteinaceous casts in the convoluted and collecting tubules.

The diagnosis was granuloma of the left kidney, due to Mucor, with subacute interstitial nephritis of the right kidney.

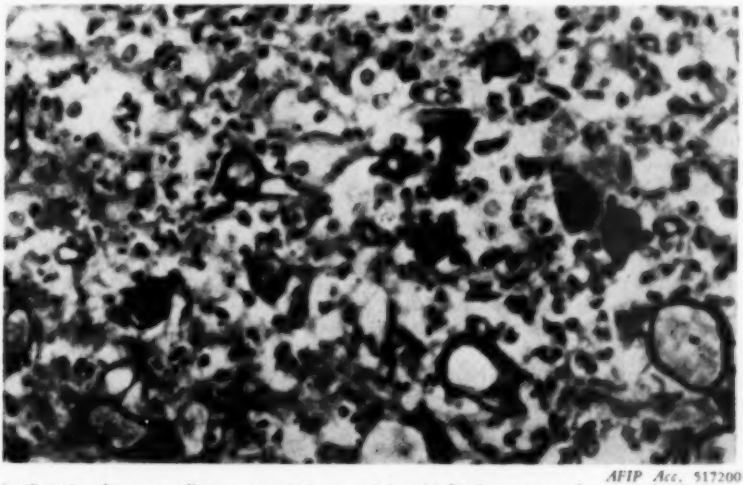
Case 2 (AFIP Accession 517200).†—A 3-month-old male Terrier had had bloody diarrhea for five days, anorexia, serous discharge from the eyes, and a rectal temperature of 102.2 F. A fecal examination revealed ova of coccidia and ascarids. The pup was given an anthelmintic and the diarrhea cleared up, but recurred in a few days. General "chewing fits" were observed at the time the pup was admitted to the hospital, and he was unable to retain food. Neurological signs consisted of unsteadiness of the hind legs and dilatation of the pupils. The pup died thirteen days after it was first seen.



AFIP Acc. 517200

Fig. 3—Gastric ulcer with marked granulomatous reaction. Cross sections of the hyphae are visible at this low magnification. x 55.

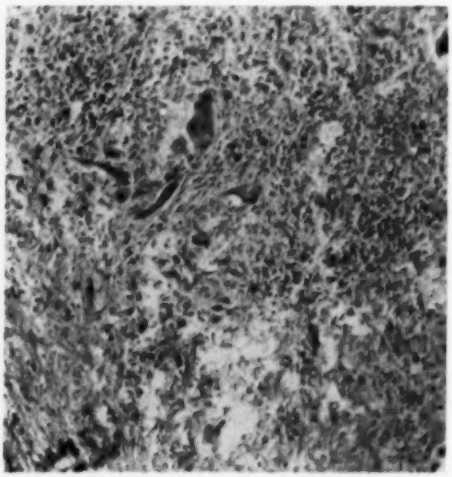
†Contributor, Dr. Mathew A. Troy, Pelham, N. Y.



AFIP Acc. 517200

Fig. 4—Gastric ulcer revealing cross sections and longitudinal sections of coarse hyphae. x 440.

At autopsy "an ulcer about ¾ inch in diameter with a raised hemorrhagic perimeter was present in the fundic region of the stomach. The anterior portion of the small intestine appeared normal, and the



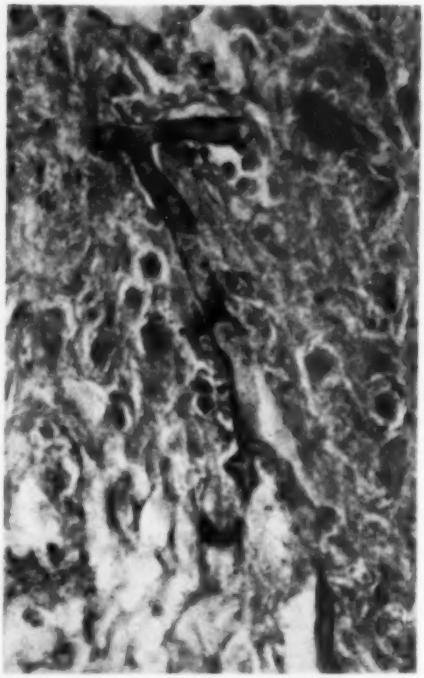
AFIP Acc. 522306
Fig. 5—Granulomatous reaction in lymph node. x 130.

posterior portion thickened. The colon appeared normal. Some congestion was present in the lungs. All of the vessels of the brain were congested and a clot was found in the falx cereberi."

Microscopic examination revealed a large ulcer of the stomach wherein the mucosa was entirely lacking and had been replaced by a hemorrhagic and granulomatous process extending at some points deep into the muscularis. The granulomatous process was marked by a cellular infiltration comprised mainly of neutrophils, macrophages, and multinucleated giant cells. Proliferation of fibroblasts and deposition of collagenous connective tissue were also observed. Areas of necrosis and hemorrhage were interspersed throughout this diffuse process. Many bizarre, coarse, branching hyphae were seen within this process. Frequently, cross sections of the hyphae looked like large encapsulated bodies containing spores (fig. 3, 4).

The pulmonary parenchyma around the bronchioles was somewhat consolidated and an exudate consisting of neutrophils, desquamated epithelial cells, and some macrophages was present within many of the bronchioles and alveolar ducts. The consolidated areas around the bronchioles were packed with neutrophils and macrophages.

The lymphoid follicles of the spleen were relatively devoid of lymphocytes and many of those present were immature. Numerous plasma cells were observed at the periphery of the follicles. A focal inflammatory reaction was seen in the adipose tissue immediately adjacent to the pancreas, although the organ itself appeared normal. No significant lesions were seen in the eyes, gastrointestinal tract, or urinary tract.



AFIP Acc. 522306
Fig. 6—Coarse, branching, nonseptate hyphae in lymph node. x 760.

The architecture of the cerebral and cerebellar cortices had been completely disrupted by multiple, diffuse granulomatous lesions. These areas of granulomatous change were characterized by degeneration of nerve cells, accumulation of lymphocytes and other mononuclear cells perivascularly, and prominent proliferation of astrocytes. Macrophages and some neutrophils were also observed in these areas. The etiological

agent of this diffuse multiple encephalitis was not seen in the sections.

The diagnosis was mycotic gastric ulcer caused by Mucor with a diffuse granulomatous encephalitis, cerebral and cerebellar cortex.

Comment.—The tissue sections from this case were referred to Dr. C. W. Emmons. It was his opinion that the structures observed in the gastric ulcer were typical of Mucor as seen in tissues. The coarse branching hyphae were like those of *M. corymbifer*.

Case 3 (AFIP Accession 522306).‡—A 1-year-old heifer was noted by the inspector in a slaughterhouse to have extensive caseocalcareous lesions in the bronchial and mediastinal lymph nodes. No other lesions were evident grossly. Histologically, the architecture of the nodes was completely disrupted by extensive necrosis, scarring, and secondary calcification. The extreme cellularity of certain areas was due to the large numbers of lymphocytes, plasma cells, macrophages, and numerous multinucleated giant cells of the Langhans type. The coarse, branching, nonseptate hyphae characteristic of Mucor were uniformly observed throughout the section and frequently within the giant cells (fig. 5, 6). The engulfed hyphae were not readily visible in the hematoxylin and eosin preparations, but were beautifully demonstrated in sections prepared by the periodic acid-Schiff method. A Ziehl-Neelsen preparation failed to reveal the presence of acid-fast organisms.

DISCUSSION

In each of the three cases presented, a fungus with coarse, branching, nonseptate hyphae, morphologically compatible with Mucor, was found on microscopic examination. The fungus was incorporated in a marked granulomatous reaction and was actually being phagocytized by multinucleated giant cells. In none of these cases, even in specially stained sections, was there evidence of another etiological agent which could have been responsible for the severe reactive changes. It is difficult to conceive of a contaminant so intimately associated with the violent pathological process described, or growing inside of the blood vascular system as observed by Gregory, Golden, and Haymaker.[9] Therefore, the concept that Mucor is a contaminant in these three cases is unacceptable. Rather, the histological and clinical pictures confirm the pathogenicity of Mucor in the animal species described.

‡Contributor, Dr. C. L. Davis, Denver, Colo.

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There is an apparent increase in mortality of turkey flocks due to *Salmonella typhimurium* and several persons have been infected while handling such birds. This is the leading Salmonella offender in provoking food poisoning.—*Canad. J. Pub. Health, July, 1953.*

Control of the Fowl Tick, *Argas Persicus* (Oken), on an Alabama Poultry Farm

S. A. EDGAR, Ph.D.; C. D. LITTLE, M.S.; J. F. HERNDON, M.S.

Auburn, Alabama

AN OPPORTUNITY was afforded the authors to attempt control of the fowl tick, *Argas persicus*, under farm conditions when an outbreak occurred on the farm of a Montgomery, Ala., poultryman in 1951.* An examination of the premises revealed a fairly heavy infestation of ticks in cracks, crevices, and other hiding places in four small henhouses, whereas, none were found in one unused brooder house or in the cluttered chicken yards. The owner reported that 30 hens had died in the previous few weeks. He thought that their death was due, in part at least, to the bites of the ticks because some of the birds appeared to be anemic before death. Several insecticides and roost paints had been tried over a period of several months but the owner had been unable to eradicate the ticks. However, his treatment for ticks had not been thorough. Although the occurrence of *A. persicus* is spotty and not common in Alabama, since other poultrymen likewise have experienced considerable difficulty in eradicating this tick from their farms and since there is little practical information on its control,[1] a field test was undertaken.

The chicken yards and houses of the farm under study were cluttered with old boards and other trash, a condition common to many poultry enterprises, making it a difficult place to eradicate ticks. Knipling[3] has pointed out that several chemicals can be used against fowl ticks but success depends on how thoroughly the materials are applied. In laboratory tests involving 1,401 substances,[2] dieldrin, lindane, toxaphene, chlordan, and aldrin were found to be the most promising for fowl tick control. Residual laboratory tests indicated that at 5.0 to 0.5 per cent cencentrations, except for aldrin which was tested for only one week, these four compounds may remain effective for as long as twenty-three weeks. Smith[4] reported on the successful control of the fowl tick under field conditions with aldrin, chlordan, toxaphene, and lindane. In view of these findings and reports of other investigators, sulfur dust, aldrin, lindane, and heptachlor were used in the field trial reported here.

In the first attempt at control, sulfur, aldrin, lindane, and heptachlor were used in different houses. One application of each compound was applied. Sulfur was sprinkled on the litter, roost poles, and in nests of house 1 (200 sq. ft. of floor space) at the rate of 2 lb./100 sq. ft. of floor space. A 0.6 per cent water emulsion of aldrin was sprayed on the walls, roosts, nests, and floor of house 2 (50 sq. ft. of floor space) at the rate of 500 cc./100 sq. ft. of surface area (200 sq. ft).**

A 0.114 per cent water suspension of lindane was applied in the same manner in house 3 (225 sq. ft. of floor space) at the rate of 1,100 cc./100 sq. ft. of surface area (700 sq. ft.) A 1.0 per cent water suspension of wettable heptachlor was sprayed in house 4 (60 sq. ft. of floor space) at the rate of 800 cc./100 sq. ft. of surface area (240 sq. ft.).

All compounds were applied in the morning. Birds were allowed to remain in the house treated with sulfur, whereas, birds were kept out of houses treated with other compounds until the evening of the day sprayed. There was no clean-up of trash in any of the yards or houses, and no compounds were applied in yards since birds roosted only in the houses.

At intervals after the insecticides had been applied, the owner observed many remaining live ticks in the house treated with sulfur, a few in the house treated with aldrin and heptachlor, and none in the house treated with lindane. On Jan. 31, 1952,

From the Agricultural Experiment Station, Alabama Polytechnic Institute, Auburn.

*The writers gratefully acknowledge the assistance of Mr. Bob I. Bright, assistant county agent, Montgomery, Ala., who called this outbreak to our attention, and the coöperation of Mr. W. L. Ingram on whose farm the study was made. The writers are also grateful to Julius Hyman and Co., who furnished aldrin; to the Agricultural Chemical & Supply Co., Montgomery, Ala., for furnishing lindane and sulfur; and to Velsicol Corp., for furnishing heptachlor.

**On the surface area, which included the walls and floor, sprays were applied with a hand sprayer.

three months after treatment, the senior author searched each house carefully and found many live ticks of all stages in houses 1 and 3, which had been treated with sulfur and lindane, respectively. A few live ticks were found in house 4, treated with heptachlor, and only 2 were found in house 2 where aldrin had been used. Many dead specimens were found in cracks and crevices, particularly in those houses treated with aldrin and heptachlor.

In view of the near eradication in house 2, which had been treated with aldrin, house 1 (500 sq. ft. of surface area), which still had a heavy infestation of ticks after having been treated with sulfur, was sprayed on Jan. 1, 1952, with a 0.67 per cent suspension of aldrin applied at the rate of 800 cc./100 sq. ft. of surface area. Although the walls, roosts, and floor were covered as thoroughly as possible with the amount applied, a few live ticks were found in this same house three months later (April 4).

The other houses that had been treated once in October, were again examined for ticks at this time. Several live ticks of all stages, were found in houses 3 and 4 treated with lindane and heptachlor, respectively; none was found in house 2 where aldrin had been sprayed five months earlier. At this time, the interior of houses 1, 3, and 4 were sprayed thoroughly with a 2 per cent water emulsion of aldrin at the rate of 1,200 cc./per 100 sq. ft. of surface area in each of houses 1 and 3, and at the rate of 800 cc./100 sq. ft. of surface area in house 4. All houses were carefully checked seven weeks later and again at six and one half and eleven months after the April 2 spraying. No live ticks were seen at any time. It is the opinion of the authors that a lapse of eleven months after spraying, especially during the warmer months of the year, is sufficient time to expect that the ticks may have been eliminated; however, in view of the nature of this tick, conclusions as to eradication can be reached only after several more months of observation.

These four compounds, especially aldrin, although applied liberally, gave no indication of being harmful to the chickens. However, birds were kept from houses until six to eight hours after sprays were applied. Although three of the compounds were not tested sufficiently to say that they are ineffective in eradicating ticks under these conditions, it is in the opinion of the authors that there is sufficient evidence indicating that aldrin can be used successfully in controlling ticks in chicken houses without moving laying hens to new quarters. Apparently, thorough spraying of the interior of houses where hens roosted was sufficient to control ticks, even though there may have been roosts in the cluttered yards that were not sprayed. To be effective, aldrin must be applied thoroughly to all surfaces of the house. Care must be exercised in handling aldrin and birds should not be exposed to excessive amounts of the compound.

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Antibiotics and Fungous Flora

Undesirable effects in man from administration of broad-spectrum antibiotics include glossitis, stomatitis, sore throat, proctitis, pruritus, nausea, vomiting, diarrhea, and bronchomoniliasis. It has been suggested that these effects result from the increased growth of *Candida albicans*, a fungus, which was demonstrated to be present in 100 healthy children as follows: in the mouths of 14 per cent, in the rectums of 6 per cent, and in the gastrointestinal tracts of 17 per cent.

When 21 child patients were given chlortetracyline (aureomycin) orally for eight days and when 24 were given chloramphenicol (chloromycetin®) intramuscularly or orally for ten to fourteen days, by the thirteenth day of treatment there was a marked decrease in gram-negative and gram-positive organisms in the gastrointestinal tract, with a concomitant increase in the fungous flora. However, the dangers do not seem great when the drug is administered for not more than eight days.—*J. Am. M. A., July 4, 1953.*

Observations on Lindane Poisoning in Dairy Animals

RAY E. ELY, Ph.D.; P. C. UNDERWOOD, B.V.Sc.; L. A. MOORE, Ph.D.;
H. D. MANN; R. H. CARTER, M.S.

Washington, D.C.

LINDANE HAS been recommended as a spray for dairy barns.[1,5] These recommendations include precautions to avoid contamination of the mangers and water bowls. Others[1,2,5] have reported that lindane appears in the milk when it is sprayed directly on dairy cows. No recommendations have been made for the use of lindane sprays on dairy animals for the control of barn flies. Deaths or toxic effects to young cattle have been reported when concentrations exceeding 0.1 per cent have been applied to the animals.[4,7] The concentration recommended for controlling certain external parasites of livestock ranges from 0.03 to 0.06 per cent. The preparations generally employed for residual spraying of barns for fly control ranges from 0.3 to 0.5 per cent.

This paper reports the observations of 11 cases of lindane poisoning, 5 terminating fatally, following the inadvertent spraying of dairy animals with greater than recommended concentrations.

OBSERVATIONS

A total of 240 animals were sprayed on two successive days (July 15-16, 1952) with a portable power sprayer, using a mixture of 5 lb. of isotox* per 50 gal. of water, that was prepared for the spraying of barn walls. This gave a suspension containing approximately 0.3 per cent of lindane. No information was available on the accuracy or uniformity of preparation of the insecticide solutions. The material was prepared and applied by individuals unfamiliar with the hazards involved and the precautions necessary in using this material. The amount of insecticide applied to individual animals undoubtedly varied widely. No attempt was made to cover the mangers or the drinking cups in the individual stalls while spraying. Later interrogation disclosed that the spray was applied directly to the animals in addition to the barn walls and equipment.

Symptoms of toxicity in 1 animal were reported approximately six hours after spraying on July 15 and were observed by the station veterinarian. These symptoms were not immediately attributed to lindane poisoning.

Symptoms of toxicity were observed in 10 additional animals that were sprayed on July 16. These symptoms appeared six to twenty-four hours after spraying and disappeared in ten to seventy-two hours. The symptoms ranged from mild inappetence, decreased milk production, incoördinated movements and muscular twitching, to violent convulsions, some ending in death. Of the 5 fatalities, 4 were young calves and 1 was a mature bull.

Autopsy of the 5 animals that died showed congestion of the heart, lungs, liver, and kidneys. Slight to severe bloat was observed in all of the cases. Two animals had reddish, turbid urine in the bladder with a flocculent precipitate. Two animals had abdominal visceral temperatures of 109.2 and 112.0 F. two hours and one hour after death, respectively. Body temperatures of the other animals were not ascertained.

Selected tissue samples were taken from these 5 animals for determinations of their lindane content. The concentrations of lindane in these tissues are presented in table 1.

TABLE 1—Lindane Content of Selected Tissues from Animals that Died with Lindane Poisoning

Animal	Spraying to death (hr.)	Parts per million of lindane in Liver	Kidney	Fat	Muscle	Blood
2307	12	1.4	0.5	7.0	....	....
3222	17	1.5	2.7	49.0	1.8	0.5
SX181	18	1.2	1.4	89.0	1.6	1.2
3190	43	1.5	3.0	250.0	....	....
3187	43	1.5	0.6	....	....	....

The concentrations of lindane in the livers of the animals showed negligible differences. A rapid transfer and storage in the fat is indicated by the marked increase in the lindane concentration of the fat, with increasing survival time of the animals after spraying. One animal (2307) that died twelve hours after it was sprayed had 7 p.p.m. of lindane in the fat, while another animal (3190) that died forty-three hours after it was sprayed had 250 p.p.m. of the insecticide in the fat. There is evidence that the *gamma* isomer of benzene hexachloride is also removed from the fat depots comparatively rapidly.[3,6]

Milk samples were taken from 3 animals that were known to have been thoroughly sprayed with the lindane solution. The concentrations of lindane in these samples and the time intervals between spraying and sampling are given in table

From the Bureau of Dairy Industry (Ely, Underwood, Moore) and the Bureau of Entomology (Mann, Carter), Agricultural Research Administration, U. S. Department of Agriculture, Washington, D. C.

*Wettable powder containing 25 per cent of lindane (gamma isomer of benzene hexachloride).

2. Maximum concentrations of the insecticide in the samples taken appeared on the afternoon of the first day after spraying. Samples taken fifty hours after spraying had approximately one half the concentrations as the samples taken twenty-six hours after spraying. Other studies[1,2] have previously reported that the maximum concentrations of lindane in the milk occur on the first day after spraying.

Oral intakes of lindane, in addition to that absorbed through the skin, were undoubtedly encountered by licking of the walls, stanchions, mangers, and body. In some cases, the ingestion of contaminated feed remaining in the mangers contributed to the oral intake. A sample of the hay remaining in the manger of animal 2307 at the time of death contained 8 p.p.m. of lindane.

DISCUSSION

The observations reported here emphasize the precautions that are necessary in using any insecticidal product on or around animals. The range between a recommended or tolerable dosage and one that will produce severe toxic manifestations may be small. Radeleff and Bushland[7] reported that lindane may cause poisoning in calves when applied at twice the concentrations needed for controlling certain ticks. Radeleff *et al.*[8] emphasized the narrow margin of safety with lindane used on young calves. They observed enough cases of poisoning in young Jersey calves with sprays containing 0.05 per cent of lindane to cause such a concentration to be considered as slightly above the maximum that is safe. They also reported that "no deaths or poisonings resulted from the use of 0.03 per cent sprays or dips on thousands of calves."

TABLE 2—Parts per Million of Lindane in Milk Samples from Cows Sprayed with Lindane

Time after spraying (hr.)	Animal N286	N287	N295
16	2.2	2.7	3.2
26	3.0	3.1	3.5
50	1.6	1.3	1.5

In view of the large number of animals that were inadvertently sprayed with a high concentration of lindane, it seems likely that many minor cases of toxicity were unobserved.

This paper does not minimize the usefulness of lindane for the control of insects for which it is recommended when used in the proper concentrations.

SUMMARY

A total of 240 animals were inadvertently sprayed with higher than recommended concentrations of a wettable powder containing 25 per cent of lindane.

Eleven cases of lindane poisoning were observed, 5 of which terminated fatally.

Toxic symptoms appeared in six to twenty-four hours after spraying, and disappeared in ten to seventy-two hours.

Symptoms of toxicity ranged from mild inappetence, decreased milk production, incoördinated movements, and muscular twitching, to violent convulsions that terminated fatally.

The body fat of the animals that died contained 7 to 205 p.p.m. of lindane.

Milk from cows thoroughly sprayed with a high concentration of lindane had up to 3.5 p.p.m. of lindane in the milk secreted during the day following spraying.

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Pasteurella multocida organisms were found to be the probable primary cause of nearly 1 per cent of mastitis cases in one survey.—*R. A. Packer, D.V.M., Ames, Iowa.*

An Illinois Practitioner's Survey of *Leptospira Pomona* in Cattle

M. ERDHEIM, D.V.M.

Grayslake, Illinois

In recent months, much has been said and written about the incidence of leptospirosis in farm animals, giving rise to speculation on the actual distribution of the disease in this country. Some surveys have been reported. Ferguson[1] tested blood samples selected at random from those submitted for the brucellosis test at the Ohio State Laboratory. Of 132 samples tested with *Leptospira pomona* antigen, 12 were positive; and of 152 samples tested with *Leptospira icterohaemorrhagiae* antigen, two were positive. There were no positive reactions to *Leptospira canicola*, *Leptospira batavia*, *Leptospira grippotyphosa*, *Leptospira bovis*, or *Leptospira autumnalis*. In Alabama,[2] routine testing of blood from 353 cows and 38 hogs selected at random revealed 14 cows as reactors at the 1:200 dilution, and 1 hog and 14 other cattle as reactors at 1:100.

But what should the average dairy practitioner expect to find in his own practice? A number of "weed poisoning" outbreaks had been diagnosed during the 1949 pasture season. There is now good reason to suspect that they were really cases of acute leptospirosis. Since July, 1950, leptospirosis has been frequently diagnosed in these dairy herds, and the diagnoses have been confirmed with serological tests. We were, therefore, motivated to run a routine survey in our practice to learn the possible distribution of the disease in our area, and to see if we could find a correlation between positive tests and previous clinical histories.

Therefore, those herds in our practice which were being tested for brucellosis were used in this study. The blood drawn for the official brucellosis test from August, 1952, to April, 1953, was tested also for *L. pomona*. The laboratory work was done by Dr. H. S. Bryan of the College of Veterinary Medicine, University of Illinois, Urbana. The samples were tested in the 1:10, 1:100, 1:1,000 and, in some cases, higher dilutions. When the results were returned from the laboratory, an attempt was made to check the history of every reacting

Dr. Erdheim is a practitioner in Grayslake, Ill.

TABLE I—Summary of Home-Raised Herds with No Recent Traffic

Herd	Tested	Reactors	Comments
Reg. Holstein-F.	66	3	
Reg. Hereford	49	19	Two months later — 47 tested, 12 reacted.
Reg. Holstein-F.	17		
Reg. Holstein-F.	40	3	All 3 reacted again to later tests.
Reg. Holstein-F.	27	4	See note 1.
Gr. Guernsey	20		
Reg. Holstein-F.	38	9	See note 2.
Gr. Guernsey	23		
Reg. Jersey	24		Three months later, all still negative.
Gr. Holstein-F.	29		
Gr. Holstein-F.	31	3	
Gr. Holstein-F.	28	8	See note 3.
Gr. Holstein-F.	29	3	
Gr. Brown Swiss	34		
Reg. Holstein-F.	46		
Gr. Holstein-F.	22		
Reg. Hereford	5		
Gr. Holstein-F.	14	1	
Reg. Holstein-F.	36		Group of isolated heifers in large herd.
Gr. Holstein-F.	33		
Gr. Holstein-F.	30		
H.-F.-Brown Swiss	37		
Gr. Guernsey	31		
23 herds	709	53	
SUMMARY OF HERDS WITH RECENT ADDITIONS			
Guernsey-H.F.	31	2	Brucella-positive herd — abortion storm year before.
Gr. Holstein-F.	25		
Gr. Holstein-F.	35		
Reg. Shorthorn	63		
Gr. Holstein-F.	52		
5 herds	206	2	
TOTALS			
28 Herds	915 Head		55 positives

Reg. = registered; Gr. = grade; Holstein-F. = Holstein-Friesian.

Note 1.—One reactor came from a herd which had suffered a severe outbreak of leptospirosis in July, 1950, followed by another outbreak fourteen months later.[3] This cow had shown no clinical symptoms in the new herd. Another reacting cow aborted a 7-month fetus in December, 1951. She was rebred and delivered a normal calf in February, 1953. However, as she went into her eighth month of pregnancy, she became stiff in all limbs and carried her head extended—a tetanus attitude. She seemed to respond to penicillin therapy—3,000,000 units daily for three days, but had to be re-treated with penicillin both before and after parturition. She is still somewhat stiff but is milking heavily and appears normal in every other way.

Note 2.—This herd has been on an artificial breeding program for a number of years. The conception rate was somewhat below average for the technician. There were some retained placentas, but not enough to cause concern. One of the reacting animals had aborted and was bred at the first following heat period. She aborted a 3-month fetus to this conception. She then was not bred at the first heat period but eventually calved normally to a subsequent breeding. This farm adjoins a farm with a lowland pasture where the herd had gone through an outbreak of "weed poisoning" in the fall of 1949, which we now suspect was leptospirosis.

Note 3.—The owner of this herd assured us, somewhat nervously, that none of the reacting animals had ever shown suggestive symptoms. He had just received the herd's negative brucellosis report and could see no point in looking for any other kind of trouble. He remained uncoöperative, but we know that the bull he bought the year before, and which now was positive at the 1:1,000 dilution, came from a farm adjoining the farm which had outbreaks in 1950 and 1951; farms also with adjoining lowland pastures.

animal for any suspicious symptoms. This was the most difficult part of the study since owners and managers were often not coöperative. They apparently associated the questions with the brucellosis test and feared the possible loss of animals with a history of "sickness."

In this report, the herds are divided into two groups: (1) those with no traffic in the past year, *i.e.*, no purchased additions and no animals returned from shows; and (2) those in the process of being built up by purchased animals.

DISCUSSION

While too few herds were studied to draw conclusions, it is evident that one may expect to find a number of reactors to leptospirosis even in the best managed herds. What these reactors signify is not clear. They apparently had never shown clinical symptoms of disease although some had aborted. Also, if these reactors had been undetected cases at one time, they apparently had not caused widespread infection in the herds. It may be that leptospirosis resembles brucellosis in that reactors and suspects may be carried in a herd for a considerable length of time without causing trouble, then suddenly some factor makes the cases acute. Obviously, much more work needs to be done in this field.

SUMMARY

1) Twenty-eight well-managed herds which were being tested for brucellosis on the eradication program were also tested for *Leptospira pomona* with the agglutination-lysis test.

2) Of 28 herds tested, 10 (36%) had reactors to *L. pomona*.

3) Of 915 head tested, 55 (6%) were positive to *L. pomona*.

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Type E and Fish-Borne Botulism.—In the past twenty years, 10 known episodes of type E botulism have been recorded, involving 41 persons of whom 13 died. Seven occurrences were in North America: three in British Columbia, two in New York State, one in California, and one in Alaska. Some form of fish or marine mammal was implicated in all but one of the outbreaks. —*Canad. J. Pub. Health, July, 1953.*

Reports on Dwarf Research

Scientists from 15 states discussed the subject of dwarfism in cattle at Kansas City recently. Dwarfism is common to all beef breeds and to some dairy breeds. Geneticists believe it is a simple recessive gene which will appear in 1 of 4 calves when carriers are mated. Three of the 4 calves will be normal but 1 of them will be a carrier. It is estimated that if all herd bulls were carriers, the incidence of dwarfism would stabilize at about 14.6 per cent. If half the bulls were carriers the incidence would be 4.4 per cent. If only 20 per cent of the bulls were carriers the incidence would be less than 1 per cent.

Drs. L. N. Hazel and M. A. Emmerson of Iowa State College reported that x-ray research on dwarfism revealed that the loin vertebrae were distinctly compressed. If more extensive research substantiates this observation it may prove to be a clue for identifying carriers.

The profilometer, which attempts to identify carriers by profile peculiarities, has been extensively studied in California and other western states. Some are optimistic while others are skeptical of its value. Purdue University scientists report the impression that the dwarf calf has many characteristics of cretinism, which occurs in man, caused by underactivity of the thyroid gland. However, when the thyroid activity of 25 dwarf calves was measured, 11 were normal and several more probably normal. When thyroid hormones were administered to 2 dwarf calves, their appetite improved and they gained in fat and general condition but there was no improvement in their skeletal or muscular growth. The effect of male hormones was quite similar.

Dr. L. C. Ferguson of Ohio State University reported on blood antigen research. As yet, he has found no correlation between the genes of dwarfism and cellular antigens. Nutrition was not considered a factor in dwarfism.—*Hereford J., Sept., 1953.*

Treatment of Tetanus

Heroic doses of tetanus antitoxin may be without value once the toxin is fixed in the central nervous tissues. Therefore, the following technique was carried out in 6 human cases, 4 of which recovered:

1) At least 250,000 units of antitoxin should be given immediately, half of it intravenously.
2) Early tracheotomy should be performed as a routine measure.
3) Every six hours, 500,000 units of penicillin and 0.5 Gm. of streptomycin should be given.
4) Early and wide excision of the wound, if one is present, should be made.
5) Continuous intragastric feeding should be administered.
6) Continuous intravenous drip of gallamine triethiodide should be administered.
7) Minimal sedation with short-acting barbiturates, such as pentothal, should be used.

It may be necessary to continue with the tracheotomy tube and the intravenous drip two or more weeks.—*J.Am.M.A., Aug. 1, 1953.*

Penicillin or Tetanus Antitoxin

When asked if penicillin given promptly would fully prevent tetanus, two authorities said "no." A case is cited of a child who died of tetanus, although given multiple large doses of penicillin promptly. Penicillin is advised locally or systemically in conjunction with tetanus antitoxin or toxoid, or both, if treatment is delayed. The dose for man should be increased from 5,000 or 10,000 units to 20,000 or 40,000 units of antitoxin. Even in those previously vaccinated with tetanus toxoid, a booster dose of toxoid may be accompanied by tetanus antitoxin. Under ordinary circumstances, a booster dose of toxoid should produce adequate protection for as long as ten years following basal immunization. However, the toxoid should be repeated at each exposure. —*J. Am. M. A., March 28, 1953.*

Fatalities Following Antibiotic Therapy

Three human fatalities after muscular injections of 400,000 units of procaine penicillin G and 1 Gm. of dihydrostreptomycin twice daily for three to six days are recorded. Diarrhea or fever commenced on the third to ninth day following the first administration, and death occurred on the fourth to eighth day after the onset of symptoms. Death appeared to be due to a toxemia, not a septicemia. Complete autopsies and bacteriological cultures were made on each case. Severe enteritis was the cause of death in each, with abundant pure growth of hemolytic *Staphylococcus aureus* in bowel cultures. In two other cases, antibiotics were discontinued promptly at the first sign and fluid and systemic therapy resulted in recovery. The concept that such complications are due to oral administration of antibiotics causing suppression of normal intestinal flora and thus permitting pathogenic overgrowth of intestinal staphylococci appears to be an oversimplification. A direct stimulation of the staphylococci by antibiotics must be considered.—*J. Am. M. A., Sept. 12, 1953.*

Antibiotics for Skin Infections

Seventy-four human patients with stubborn skin infections, mostly staphylococcic, were treated with an ointment containing 5 mg. of neomycin and 250 units of bacitracin per gram of ointment. All bacterial strains isolated were highly susceptible to a combination of these antibiotics. Clinical effects were satisfactory in 73 per cent of the cases, slight in 15 per cent, and aggravated by the ointment base in 5 per cent.—*J. Am. M.A., July 4, 1953.*

Sand-Laden Liver in a Pig

In September, 1949, the abdominal viscera of a sow from a Johannesburg abattoir were examined. The liver was enlarged with a slightly yellow tinge. Several large firm, elliptical cul de sacs were evident on the visceral surface, the largest being 10 in. long and 6 in. in diameter. These were found to be filled with sand, totalling 24 lb. 6 oz. when air-dried. The animal had a double ductus choledochus, with one branch opening into the pylorus. Presumably, the sand was forced into this duct by the gastric contractions. The author was unable to find any other such case described in the literature.—*J. of South Africa, March, 1953.*

Research at Oklahoma A. & M. College has proved the ant, as well as the snail, to be a host for the lancet fluke which affects cattle, sheep, and man.—*Bull., Nat. Soc. M. Res., March-April, 1953.*

Rabies Antiserum from Horses

There has been a re-awakening of interest in the use of rabies antiserum. Recently, 2 Alabama children died of rabies despite prompt and intensive vaccination with 14 Semple treatments. The incubation period was seventeen and nineteen days, respectively, much too short for the development of immunity. Therefore, antiserum from horses and from rabbits was made available. It would be premature to rely on serum alone but if used early, and if it prolongs the incubation period, there is more time for active immunity to be developed from the vaccine.

It may also make it possible to reduce the number of vaccine treatments from the usual 14 to perhaps seven or less, thus reducing the risk of postvaccination paralysis. It is especially valuable if the bite is in the face, arms, or hands. The risk of developing rabies is 72 times greater in face bites than in leg bites and 5 times greater in hand bites than in leg bites. Of 32 patients treated with antiserum from horses, 3 had severe serum reactions, 2 moderate reactions, 3 mild reactions. This is a better record than with rabbit antiserum and considerably better than with sheep antiserum.—*Pub. Health Rep., Aug., 1953.*

Rats are Rabies Virus Carriers.—Of 13 rats captured in Paris sewers, 3 were carriers of rabies virus. The virus was successfully passaged in laboratory rats, mice, and guinea pigs.—*Vet. Bull., July, 1953.*

Rabies in the Mongoose in Puerto Rico

The mongoose is not native to the Caribbean but was imported from India to Puerto Rico in 1870 to destroy rats in cane fields. (The rat population remains as great as on islands where it was not released.)

The mongoose has a body 14 to 17 inches long and weighs about 1½ pounds. It has a natural fear of man and many other animals but when cornered becomes extremely vicious. When rabid, it exhibits extraordinary ferocity.

In 1950, 4 dogs and 1 cat, which had fought with mongooses, two to eight weeks previously, developed rabies. Soon afterward, 2 mongooses which attacked persons were found to be rabid. During the outbreak from March, 1950, to September, 1951, a total of 78 cases of rabies were confirmed by laboratory examination, including 37 mongooses, 18 dogs, and 23 other species.—*Ceylon J. Vet. Sci., Dec., 1952.*

Rabies developed in a young lioness which had been bitten by a stray dog. Her symptoms were hyperexcitability, severe gastroenteritis, and paralysis of the hind quarters prior to death.—*Vet. Bull., July, 1953.*

Wolves—Reservoir for Rabies Virus

In Yugoslavia, wolves and foxes form an important reservoir for rabies. In 1950, 47 people were infected directly by wolves. It is advocated that all dogs be vaccinated and all wolves eradicated. Foxes, being less aggressive, are not so important.—*Vet. Bull., Aug., 1953.*

Rabies in Racoons.—Florida reports a spreading epizoötic of rabies in the racoons of that region. The population of these animals in the South is at an all-time high, possibly due to the fact that the price of raccoon hides is at an all-time low.

Aureomycin's Name Changed to Chlortetracycline.—The antibiotic long known as "aureomycin" was given that name because of its golden color. Now, by act of Congress, the name has been changed to chlortetracycline to represent its chemical formula.

Flies Transmit Swine Erysipelas.—In Germany, an experiment revealed that flies (*Stomoxys calcitrans*) were able to carry erysipelas infection from mice, liquid cultures, and from the flesh of diseased pigs to healthy pigs which later developed characteristic "diamond lesions" at the point where they were bitten. The flies remained infectious for some time.—*Vet. Bull., June, 1953.*

Sulfapyridine was ineffective for the cure of artificially produced swine erysipelas infection in mice.—*Vet. Bull., June, 1953.*

NUTRITION

Photosensitization in Cattle

In a herd of 150 grade and purebred Herefords, 75 were affected with photosensitization. The first signs were usually noticed about three days after the cows were turned on a pasture containing Bermuda grass, crab grass, red clover, bull nettle, and several other weeds. Lantana and St. Johnswort were absent.

Skin sloughing commenced six days after the first signs. The urine of the affected cattle was reddish brown but their appetites remained normal. In some, the skin was dry and hard but did not slough. In 5 animals sloughing extended from the back over the sides and to the muzzle. The nictitating membrane of 5 was completely sloughed in one eye, making them partially blind. Whether the injury was due to primary photosensitization, caused directly by material ingested, or was hepatogenic, caused by injury to the liver from products of chlorophyll digestion, was not determined.

Photosensitization was reported in 1948 three to five weeks after a frost when cattle ate the deteriorated moldy grass.—*Georgia Vet., Sept.-Oct., 1953.*

Cattle Poisoning in Georgia

The Tifton Diagnostic Laboratory reported many recent cases of poisoning in cattle. Bracken poison caused 14 deaths and prussic acid accounted for three. However, arsenic was responsible for more than 150 poisonings and 50 deaths. In one herd of 339 cattle, 66 were ill and 37 died. Not until two days after the first death was it discovered that the 30 cattle had consumed the fluid out of a dipping bath, which contained a solution of Cooper's arsenical dip. The arsenic content of the livers of 7 dead animals was from 6.2 to 48.5 γ per gram of liver tissue. Even at that late date, treatment of the sick animals with 9 cc. of British antilewisite (BAL) intramuscularly every four hours apparently was a factor in saving 29 animals.

In another herd, 250 cattle were pressure-sprayed with Cooper's arsenical dip solution, which supposedly contained ½ gallon of dip to 60 gallons of water. The second animal to die was found to have 10 γ of arsenic per gram of liver. The 35 other affected animals were also treated with BAL and recovered, although a few developed arsenical dermatitis.—*Georgia Vet., Sept., 1953.*

Vitamin C in Bracken Poison

When a steer was fed a mixture of hay and dried bracken and 2 sheep were fed almost entirely dried bracken, the steer died in forty-four days, sheep H in 125 days, and sheep K in 289 days. Sheep K had received vitamin C subcutaneously for the first fifty-seven days, after which poisoning did not appear for 153 days. However, while there may be an increased destruction of vitamin C in the terminal stages of bracken poisoning, there is no evidence of a simple deficiency resulting from bracken feeding. The leukopenia reported in cattle also occurs in sheep. Sheep apparently can tolerate bracken feeding much longer than cattle, but in the terminal stages of the poisoning they also show a deficiency of platelets and a progressive fall in erythrocytes and hemoglobin.—*Brit. Vet. J. Aug., 1953.*

Chlortetracycline and B_{12} Fed to Dogs.—The increased growth in dogs given vitamin B_{12} or chlortetracycline (aureomycin) with a complete purified diet was attributed solely to the increased fat deposition.—*Vet. Bull., June, 1953.*

Vitamin A for Hyperkeratosis

Both in the field and in controlled experiments, the feeding of hyperkeratosis-producing material caused a severe depression on the concentration of vitamin A in the blood plasma. When vitamin A (25,000 I.U. per day) was fed with the hyperkeratosis-producing feeds, it retarded but did not prevent fatal termination. However, if the toxic material was removed in the early stages of the disease, supplementation with vitamin A was beneficial. The deter-

mination of vitamin A in the blood plasma is helpful, both in diagnosing hyperkeratosis and in following the progress of a case.—*J. Dai. Sci., June, 1953.*

Saline Drinking Water for Animals

Definite tolerances for minerals in the drinking water of farm animals have not been established, but for man, the total solids permitted is 1,000 p.p.m. In many areas, water of this quality is not available for animals. While they will not voluntarily choose highly saline water, animals can be forced to adjust themselves to levels much higher than are recommended.

Sheep seem to be more tolerant to saline water than cows, and cows more than horses or pigs. Cattlemen consider 2,500 p.p.m. the maximum permissible. However, in Australia where good water is unobtainable, the upper limits considered safe are: for poultry 2,800 p.p.m., swine 4,200, horses 6,400, beef cattle 10,000, and for adult sheep 12,900 p.p.m. Lactation and reproduction are generally the first functions to be disturbed by highly saline water. —*Nutr. News Bull. July, 1953.*

Use of Inedible Animal Fat in Feed.—When 4 per cent of inedible animal fats, stabilized with antioxidant, was added to the basal ration of the bitch, it increased the lactation, and the pups gained more rapidly. The fat also increased the vitamin A stability of rations stored at room temperatures. The addition of fats to the ration of cattle and swine also is beneficial. As a result, producers of commercial feed are rapidly adding fats to their feeds.—*Am. Meat Inst. Found., Aug. 17, 1953.*

Block versus Loose Salt for Dairy Cattle

In a three-year experiment on three types of pasture, one group of dairy cows had access to loose salt, another group to block salt. In seven of nine instances the cows ate more loose salt than block salt. However, the lower intake of block salt was adequate to meet the needs for lactation. In one year when no other source of salt was available, the cows ate twice as much loose salt as block salt but the excess was apparently luxury consumption.—*J. Dai. Sci., July, 1953.*

Growth Factors for the Foal

Feeding vitamin B_{12}, chlortetracycline (aureomycin) and penicillin did not increase the rate of growth of foals, aged 6 to 8 months, when fed in the amounts which increased the growth rate of pigs.—*Vet. Bull., July, 1953.*

Simple Minerals for Cattle

At Washington State College, it has been determined that only in cobalt- or iodine-deficient areas is it necessary to feed more complex minerals than simple steamed bone meal and ample salt. Analyses of bone meal samples from several states and foreign countries were found to contain adequate amounts of all the major minerals needed by dairy cattle and all but the above two minor or trace minerals. Fancy mineral mixtures may cost up to $240 a ton, whereas bone meal is about one-third that amount. Iodine can be provided as iodized salt.—*Farm J., Oct., 1953.*

Soft Phosphate in Swine Rations

In recent experiments, Pennsylvania State College determined that soft phosphate, with colloidal clay, was the equivalent of U.S.P. grade dicalcium phosphate in the ration for pigs. There was no significant difference in the weight gains, feed requirements, or phosphorus contents of the tissues. There was a highly significant difference in the fluorine content of the bones but the increase, when soft phosphate was fed, was not sufficient to cause fluorine poisoning.—*Am. Feed Mfr. A. Nutr. Abstr., Sept., 1953.*

Fate of Intravenous Iron in Cats

An investigation in young cats with iron deficiency revealed that most of the iron administered intravenously was deposited in the liver by the second or third day after the last injection. When amounts greatly exceeding therapeutic doses were injected, some iron was found in the lungs and occasionally in the spleen but the kidneys were always free. Since therapeutic utilization is greatest when the iron passes through the liver, intravenous administration seems to provide an efficient and rapid method of compensating for deficiency.—*J.Am.M.A., July 11, 1953.*

EDITORIAL

A Little Knowledge—A Dangerous Thing

In this atomic age many have warned that man's ingenuity for invention is greater than his capacity for restraint; thus he might create a device that would lead to his own destruction.

How true this could be is now being illustrated, in a minor way, not with chemical or mechanical devices but with a pathogen.

Man has controlled many pests simply by fostering their natural enemies. A fatal contagious disease is one agent that can be used for this provided, of course, that only the pest is susceptible. Thus, after twelve years of experimenting, a fatal virus disease affecting that species only was seeded among the destructive hordes of wild rabbits in Australia. The results were highly satisfactory — full knowledge had apparently been applied. However, less knowledge was less wisely applied when a citizen of Europe, where wild rabbits can scarcely be classified as serious pests, somehow secured some of that virus and seeded it in the few bunnies that molested his premises. This was a dangerous thing — now the valuable domestic rabbit industry of Europe is threatened.

The agent used was the virus of myxomatosis, a disease the visible symptoms of which are inflammation of the eyes, swelling of the head and other parts, and multiple fibromas in the skin. It is a loathsome disease never before recognized outside of the Americas and almost always terminating in death in one to two weeks. It is spread by mosquitoes and other insects but on a few occasions has jumped hundreds of miles, suggesting that birds must also be vectors.

In the three years since the disease was introduced in Australia, it is estimated that the rabbit population has been reduced by 90 per cent, a figure it may not much exceed since a species probably can never be completely eliminated by a virus. Invariably, a few will survive and eventually may repopulate the area. Nevertheless the campaign has made available pastures sufficient to graze an additional 10 million sheep in the affected area.

The progress of the epizoötic in Europe is reported, like a continued story, by the monthly *circulaire* of the Office International des Epizoöties, Paris. The July 7, 1953, *circulaire* reported that in October, 1952, myxomatosis had for the first time appeared in France; its origin was then unknown but it was spreading rapidly among wild rabbits in the central and northern regions. The August *circulaire* revealed that "A landowner wishing to destroy the wild rabbits on his premises, used the *Molitar myxomae* virus for the purpose." By September the disease was reported to have been diagnosed positively in Belgium and tentatively in Germany.

Since learning about this epizoötic in Europe, rabbit breeders and manufacturers of rabbit food in this country have been demanding that federal action be taken to prevent importation of the disease.

All of this furore apparently has resulted because one person used a drastic scientific means to destroy a relatively minor irritant without knowing or considering the consequences. *Time Magazine* (Aug. 10, 1953) reported the person as being an educated man, a pediatrician, who at first was so proud of his achievement that he made a report on its early success.

The mortality caused among European rabbits has not been reported. An attempt is being made to save the domestic rabbit industry by immunizing with a vaccine, derived from the virus of the similar, but benign, Shope fibroma of rabbits. As yet, there has been no report on the results of this vaccination.

French authorities warn that myxomatosis may become a panzoötic and they urge that immediate measures be considered in an attempt to prevent this. Truly, a little knowledge can be a dangerous thing.

In Handling Livestock, "Easy Does It"

The agricultural research department of Swift and Company is to be congratulated on their recent pamphlet, "Easy Does It." This cleverly illustrated 16-page brochure

points out the various ways that animals can be injured in transportation or at home and how most of these injuries could be avoided. One out of 10 hogs and 1 out of every 15 cattle arrive at markets with bruises which cause heavy losses. Everyone concerned with the livestock industry could profit by reading this pamphlet.

Eradication versus Vaccination for Foot-and-Mouth Disease

Little is being said about the foot-and-mouth disease (*aftosa*) situation in Mexico except that there is a wide difference of opinion between Mexican and United States officials as to how the recent outbreak should be handled. Eradication procedures were halted by the Mexican government. Vaccination is delayed because of the unavailability of fresh vaccines, as well as the unwillingness of U. S. officials to use vaccination as the only weapon.

Authorities point out that (1) *aftosa* has never been eradicated with vaccines unless accompanied by slaughter of diseased and exposed animals; (2) it is much less expensive to eradicate and live without the disease than to vaccinate and live with it.

In 1952, western Europe, with a livestock population about equal to that of the United States, suffered losses from *aftosa* estimated as high as $9 million.

In France, 6 million animals were vaccinated between Sept. 1, 1951, and Sept. 30, 1952, yet 314,605 outbreaks occurred involving 5,221,377 cloven-footed animals. In Great Britain where eradication but no vaccination was used, 578 outbreaks involving about 35,000 animals occurred during a similar period.

The cost in France, including deaths, abortions, and loss of meat and milk production, was about $285 million. This did not include the vaccination cost which was about $8.6 million. In Great Britain the total cost, including indemnities, was only $8.4 million.

An additional source of concern to authorities is the constant threat of the development of virus variants which seems to occur from passage of the virus through the same or different species during an outbreak.

Because of these variants, plus the proved low value and short duration immunity from vaccination, the immunizing method is considered incapable of eradicating the disease.

It is unfortunate that many gained the impression from the previous Mexican campaign that vaccination was an acceptable substitute for a vigorous eradication program. Vaccination might better be considered as merely a psychological weapon when properly used. Actually, it was necessary to resort to slaughter since *aftosa* sometimes appeared after vaccination; in one instance, after four vaccinations.

With the virus so readily carried by birds, insects, animals, and various contaminated objects which many travel great distances, foot-and-mouth disease is truly an international problem and as such requires our top priority attention. However, as with so many other international problems, there may never be a complete meeting of the minds on how it should best be handled.

Rendering Plants Pose a Problem

The National Renderer's Association, Washington, D. C., has issued a distress call which is of great significance for those interested in the control of communicable animal diseases. More than 45 rendering plants have been forced to close recently because of unprofitable prices for tallow and grease, hides, and animal proteins. Most of the closed plants have been in rural areas where long collecting routes, the high cost of trucking, and reduced income have made it impossible for them to continue operating. Others are refusing to make long calls for dead animals; when they do, they will only accept carcasses which can be processed profitably.

Obviously, this situation creates potential health and sanitary hazards. It necessitates the burning or burying of many carcasses which, if done at all, may be carried out in a haphazard, inefficient manner. This is a hazard, especially with such animals as poultry and pigs, where there is a temptation to toss them into a roadside ditch or into a ravine, creek, or river, where they may be carried to other premises and set up new foci of infection. Rendering trucks, when not properly supervised or operated, have often been accused of carrying infection from one premise to another, but this undoubtedly is the lesser of the two evils.

This economic squeeze has been created by the decline in the demand for inedible animal fats, largely due to the replacement of soap by synthetic detergents and also

by the reduced value of hides because of synthetic substitutes for leather.

From the esthetic standpoint, the neglect of a decomposing carcass may be more objectionable during warm weather, but this decomposition will destroy the viruses and most pathogenic bacteria, the chief exceptions being spore-bearing organisms and perhaps *Erysipelothrix rhusiopathiae* which sometimes survives for a considerable period in a decomposing carcass. However, an unburied carcass may be more dangerous during cold periods when the organisms, especially viruses, may be preserved for considerable periods in the chilled or frozen tissues. This problem merits the prompt attention of sanitary officials.

The Epizootic Score Card

VESICULAR EXANTHEMA

On Sept. 21, 1953, vesicular exanthema was present in nine states: California, Texas, Florida, and six northeastern states. The most disturbing problem is the occasional appearance of the disease in swine recently shipped from the eastern hog belt where the disease can not be found. Apparently, they somehow pick up infection at the market where loaded. About 31 per cent of the 14,000 feeders using garbage are now cooking it, and 37 per cent are being routinely inspected semimonthly. The Bureau of Animal Industry requirements for cleaning and disinfecting vehicles have been changed to require this before but not after shipment, except in special cases.

ANTHRAX

In the first eight months of 1953, 184 outbreaks were reported in 25 states with a death loss of 310 cattle, 111 swine, 51 sheep, 1 horse, and 86 mink. The mink died after eating infected meat, all on one Minnesota ranch. The heaviest infection was in Illinois with 45 outbreaks (37 in July) and chiefly in two counties where anthrax had not been reported previously. In an effort to prevent a recurrence of the 1951-1952 epizoötic, two steps taken by the BAI are: 1) New regulations prohibit the importation of bone meal unless heat treated, and 2) requirements in the production of anthrax bacterin have been made more strict. More test animals must be used and they must be held longer (sixty days) under observation.

BLUETONGUE

This virus disease of sheep has, in the summer of 1953, been widespread in California and Texas. In Arizona and New Mexico it is confined to the Indian reservations. The Bureau of Animal Industry is making extensive surveys on its extent and on the insect vectors which may be involved. A vaccine to be released in infected areas has been prepared from the two, possibly three, strains of virus so far identified in this country.

SCRAPIE

No new cases of this virus neurosis of sheep, which first appeared a year ago, have been reported since February.

In California and Illinois all affected and exposed sheep were destroyed. One entire flock, of the five in Ohio, was slaughtered; in the other four, only the affected animals were destroyed, the balance of the flocks being under strict state and federal quarantine.

Leptospirosis Vaccine Coming Soon

The leptospirosis vaccine, originated by Drs. Baker and York at Cornell University, is expected to be marketed soon by the Pitman-Moore Company, Division of Allied Laboratories. This vaccine will be available to veterinarians only. It is recommended for herd replacements, when assembling commercial herds, for animals shown at fairs, and when the disease appears in the neighborhood. In affected herds, it is recommended that all animals be blood-tested and if less than half of them react, the nonreactors as well as all new calves and replacements be vaccinated. —*C. J. York, D.V.M., Indiana.*

Go Slow, a Timely Warning

Dr. William H. Feldman, of the Mayo Clinic, says: "So far, there has not been discovered any effective chemotherapeutic substance which is devoid of potential toxicity. Perhaps no drug administered orally or parenterally is entirely without some hazard. . . . The safety of chemotherapeutic procedures is best assured when the responsibility is shared by those who, by training and experience, are aware of the hazards, and are capable of recognizing untoward reactions early in their inception."

CURRENT LITERATURE

ABSTRACTS

Erythrocytic Inclusion in Swine

Four pigs in a single herd were found to be harboring an unusual type of inclusion in cells of the erythrocytic series of the blood. The structures were associated with an acute hemolytic anemia attributed to Eperythrozoon infection in 2 of the animals and apparently unassociated with any pathology in 2 others. The inclusions appeared primarily as coccoid clusters within the erythrocytes. Also numerous were large ovoid structures up to 4.7 μ in size and containing a large vacuolated central area. As many as 20 per cent of the erythrocytes were affected. Using the Prussian blue test, the bodies gave a positive reaction for iron. Experimental transmission to other swine could not be accomplished. The similarity of these inclusions to those observed in human patients affected with congenital familial anemias and acquired hemolytic anemias of unidentified etiology is discussed.—[*Earl J. Splitter: Observations on an Erythrocytic Inclusion in Swine. Am. J. Vet. Res., 14, (Oct., 1953): 575-577.*]

Transmission of Parasitic Worms Between Sheep and Cattle

Calves were much more susceptible to the stomach worm, *Haemonchus contortus,* of bovine origin than to the same species of ovine origin. Conversely, lambs were much more susceptible to this parasite of ovine origin than to that of bovine origin. Two other bovine nematodes, *Cooperia pectinata* and *Cooperia punctata,* were acquired in a much larger number by calves than by lambs when both hosts were exposed to them. *Strongyloides papillosus* and *Trichostrongylus colubriformis,* known parasites of cattle and sheep, proved to be adapted to cattle. The tapeworm, *Moniezia expansa,* was acquired by cattle and sheep, whereas *Moniezia benedini* was acquired only by cattle.—[*Dale A. Porter: Cross Transmission of Parasitic Worms Between Cattle and Sheep. Am. J. Vet. Res., 14, (Oct., 1953): 550-554.*]

Studies on Mink Food

This study concerns experiments designed to elucidate the problem of botulism on mink ranches. Mink were fed toxic cultures of types A, B, C, and E *Clostridium botulinum.* In the doses of toxins fed, mink were resistant to types A, B, and E but susceptible to type C. Samples of mink food prepared at a particular ranch were inoculated with toxin-free spores of types A, B, C, and E *Cl. botulinum.* In these food samples, incubated at 30 C. for twenty-four hours and subsequently tested in mice, toxin did not develop. Control beef heart medium similarly inoculated became toxic in twenty-four hours with types A and C but not with types B and E. *Macaca mulatta* were found susceptible to feeding of toxic cultures of types C and E *Cl. botulinum.* Mice were injected intraperitoneally with the supernatant fluid from samples of mink food and ground lean beef inoculated with type C *Cl. botulinum* spores plus *Lucilia illustris* fly larvae or eggs. Lean beef was found to be a much more favorable medium for type C toxin production than mink food. The role of *L. illustris* larvae has been clearly demonstrated as being a favorable factor for growth and toxin production of type C *Cl. botulinum* when the spores were experimentally inoculated into lean beef and mink food.—[*R. O. Wagenaar, G. M. Dack, and D. P. Mayer: Studies on Mink Food Experimentally Inoculated with Toxin-Free Spores of Clostridium Botulinum Types A, B, C, and E. Am. J. Vet. Res., 14, (July, 1953):479-483.*]

Effect of Transplantable Lymphomatosis on Young Chickens

Two experiments, involving 69 young domestic chickens *(Gallus domesticus),* were conducted to determine the effects of a strain of transplantable lymphomatosis upon body growth, growth of selected organs, red and white blood cell levels, differential white cell values, and body temperature. The growth of the chickens was not altered until the last quarter (48 hr.) of the experiment period. Livers, spleens, and kidneys were enlarged after the midpoint of the experiments. A definite increase occurred in white cell levels and was due to an increase of neutrophils. Levels of red blood cells, lymphocytes, and other white cell types were not appreciably altered. No significant change occurred in body temperature.—[*L. M. Julian: Effect of Transplantable Lymphomatosis (Strain RPL 16) On Young Rapidly Growing Chickens. Am. J. Vet. Res., 14, (Oct., 1953): 601-608.*]

Differentiation of Hookworm Ova in Dogs

Two types of hookworm ova were observed in routine fecal examination of dogs. In attempting to identify these ova on a size basis, the literature showed radical disagreement in ova sizes. These discrepancies are tabulated.

Hookworm ova were collected from 147 dogs

from Indiana and surrounding states during the latter six months of 1952. Only *Ancylostoma caninum* and *Uncinaria stenocephala* were found. *Uncinaria stenocephala* was observed in dogs from Ohio, Illinois, Indiana, and Kentucky.

Examination of ova in several mediums clearly indicated that *U. stenocephala* produces larger ova than does *A. caninum*. Ova from these two species were measured in the following reagents: lactophenol *(in utero)*, physiological saline *(ex utero)*, sodium dichromate, zinc sulfate, sodium nitrate, sugar, and sodium chloride. Sizes obtained were tabulated: for *A. caninum*, 55 to 74 by 37 to 43 μ; for *U. stenocephala*, 71 to 93 by 37 to 55 μ. No significant overlap of size range was found in any one flotation solution.—*[Frank A. Ebrenford: Differentiation of the Ova of Ancylostoma Caninum and Uncinaria Stenocephala in Dogs. Am. J. Vet. Res., 14, (Oct., 1953): 578-580.]*

Chemical Composition of Urinary Calculi from Range Steers

A series of 63 occluding urethral calculi from range steers were analyzed qualitatively for protein, silicon, calcium, magnesium, ammonium, aluminum, oxalate, carbonate, phosphate, and urate by methods detailed in the paper. All of the stones (except a single magnesium phosphate stone) contained protein and silicon, 20 of them nothing else. In addition, calcium was found in about one-half of the stones and oxalate in about one-third. The other components were found much less frequently. Aluminum and urate were not detected in any. Calculi from different organs of the same animal, though all containing protein and silicon, did not always contain the same minor components. Silica analysis of ten stones showed them to contain 50 to 75 per cent of silica. The significance of these observations in the etiology of calculus formation in range steers is discussed without drawing any definite conclusions. —*[Karl F. Swingle: The Chemical Composition of Urinary Calculi from Range Steers. Am. J. Vet. Res., 14, (Oct., 1953): 493-498.]*

FOREIGN LITERATURE

Cholecystography in the Dog

In studying the pathological conditions of the bile duct of dogs, the author proposes an indirect method of repair, *i.e.*, cholecystography, using the same method as in man.

A contrast substance, B-(4-hydroxy-3, 5-diiodophenyl)-phenyl-propionic acid (C_{15} H_{12} O_3 I_2), known commercially as biliselectan, was administered orally.

Twenty dogs were used as subjects for the experiment, their age, sex, and general conditions being variable.

After fasting the animal for twenty-four hours, a simple radiograph was taken. Then four tablets were administered at intervals of five minutes, and radiographs taken every hour until the desired image was obtained.

The dog was then submitted to the Boyden test, administration of two egg yolks orally. In thirty or forty minutes, a second radiograph was taken showing the form and volume of the gall bladder, thus permitting evaluation of its functional activity.

After the experiment, the author concluded that the first radiographic examination should be made twelve to fourteen hours after the ingestion of the contrast tablets, that the optimum dose was four tablets, and that it is possible to perform cholecystography in dogs as a routine procedure.—*[Mauricio Killner: Cholecystography in the Dog. Anais do V Cong. Brasileiro de Vet., Brazil (1951):277-282.]*—O. A. L.-P.

Spontaneous Erysipelothrix Rhusiopathiae Infection in Pigeons

The author reports death by spontaneous *Erysipelothrix rhusiopathiae* infection in a female pigeon and her 4-day-old squab. Typical lesions of diamond skin disease were produced in swine with the isolated strain by combined intravenous, intraperitoneal, and intracutaneous infection. Pigeons and swine were immunized with the pigeon strain against a challenge infection with *Ery. rhusiopathiae* strain of swine origin.—*[C. A. Van Dorssen and J. Donkervoet: The Spontaneous Erysipelothrix Rhusiopathiae Infection in Pigeons. Tijdschr. voor Diergeneesk., 78, (June, 1953):501-513.]*—L. V. E.

Q Fever Contracted in a Laboratory

The authors refer particularly to the clinical and laboratory characteristics observed in four cases of Q fever contracted by laboratory workers while handling infected material in Spain. Reference is made to nearly 100 similar infections observed in four laboratories in the United States.

One of the patients was a woman laboratory assistant whose only contact with infected material was while taking the temperature of the infected guinea pigs, but who probably contracted the disease by inhaling aerosols or dust containing rickettsias from the rooms where work was done with inoculated chicken embryos. She presented the most severe symptoms.

Another patient, a male laboratory assistant, worked directly with infected chicken embryos. The third patient was a young medical student who presented the mildest symptoms of the four patients.

A laboratory veterinarian was also affected and showed more symptoms than the preceding two cases. The use of chloromycetin® in this case was rapidly effective in curbing the fever and other symptoms.

The medical student and the veterinarian had only one possible source of contagion: infectious

material eliminated by dogs experimentally inoculated with Q fever. It is possible that the urine and the bloody feces of those dogs may have been infective.

Guinea pigs inoculated intraperitoneally with blood from the patients did not reveal symptoms of infection.

Complement-fixation studies definitely confirmed the diagnosis of Q fever.—[*G. Clavero, F. Perez Gallardo, and E. Valle: Four Cases of Q Fever Contracted in the Laboratory. Rev. de Sanidad e Higiene Pub., Spain, 26, (March-April, 1952):199-201.*]—O. A. L.-P.

The Failure of Estrus in Swine

The author declares that the causes of sterility of swine are apt to vary. Quite often, the cause is an alimentary deficiency. In the sandy areas in Belgium, the ration is often too rich in carbohydrates and much too poor in proteins of animal origin, especially A.P.F. The feeding of 39 sows treated in 1952 for anaphrodisia was studied. The rations of 36 of the 39 were poor in proteins and only 3 received proteins of animal origin. These observations show that insufficiency of proteins have at least a predisposing influence on anaphrodisia in the sow.—[*E. Maenhout: On the Failure of Estrus in Swine. Vlaams Diergeneesk. Tijdschr., 22 (May, 1953):97-103.*]—L. V. E.

Imported Case of Babesiosis Canis

A chronic infection of babesiosis was discovered in 7 dogs. The number of parasites in the blood remained low. Hemoglobinuria was not apparent, but serious cerebral disturbances developed in 4 of the sick animals.—[*C. J. van Nie and P. Sutmoller: An Imported Case of Babesiosis Canis. Tijdschr. voor Diergeneesk., 76, (June, 1953): 514-517.*]—L. V. E.

The Evaluation of Bovine Semen

The author discusses extensively the research carried on internationally by different investigators to obtain a process capable of offering a definite assurance in relation to the fecundization power of semen. None at the present time, he concludes, offers it definitely.

The actual trend in the studies of semen is directed toward the intricacies of the metabolism of spermatozoids. Semen presents innumerable advantages for the study of cellular metabolism. It represents an example of a tissue constituted of only one type of cells, free from each other and with proper life, not reproducing nor multiplying, but constituting a regular suspension of known density. Spermatozoa possess two definite biological functions—fertility and motility—that not only distinguish them from other cells, but also place at the disposal of the researchers two definite and specific criteria to be observed in metabolic insemination.

It was the work on artificial insemination, with its economic repercussion, that started the interest of such large number of investigators on the studies of semen, especially after the discovery of its easy obtainment by means of the artificial vagina.—[*Joao Soares Veiga: Recent Progress in the Examination of the Bovine Semen for the Evaluation of its Fertilizing Capacity. Anais do V Cong. Brasileiro de Vet., Brazil (1951):135-151.*]—O. A. L.-P.

BOOKS AND REPORTS

Protozoology

This is the newest book on protozoölogy from the pen of an American author. Although dealing largely with the free-living Protozoa and written from the standpoint of a protozoölogist who is interested in the fundamentals of the biology of these microörganisms, the book will have an appeal to students of human and veterinary medicine who are interested in the Protozoa as disease-producing agents.

Following descriptions of the general morphology, reproduction and life cycles, and classifications of the Protozoa, which take up about 100 pages, the author gives a comprehensive classification, following a discussion of classifications previously proposed, of four subphyla, namely, (1) mastigophora, (2) sarcodina, (3) sporozoa, and (4) ciliophora, down to classes, orders, suborders, and families, each of the subphyla being discussed in a separate chapter. The classification covers nearly half of the book. This is followed by chapters on the physiology of, and heredity in, the Protozoa.

Of special interest to veterinarians, and even more so to medical men, are the last five chapters of the book dealing with the host-parasite relationships, Protozoa of the digestive and urogenital tracts, the blood flagellates, malaria, and immunity and resistance, presented largely from the human medical slant.

The book is readable, profusely illustrated, and should make a valuable addition to a collection of books on microparasites.—[*Protozoology. By R. P. Hall. 682 pages. Prentice-Hall, Inc., New York, N. Y. 1953. Price $13.35.*]—BENJAMIN SCHWARTZ.

The Physician in Atomic Defense

The book consists of three sections. Section I deals with atomic theory, basic physics, and atomic bombs. Section II includes the clinical, biological, pathological, and therapeutic aspects of atomic warfare. Section III discusses the organization of the medical department for atomic defense.

This book might be a helpful reference to those physicians who are especially interested in the medical aspects of atomic warfare or nuclear energy and/or who have civil defense responsi-

bilities in this field. It briefly reviews the physical concepts of matter, the structure of the atom, the significance of radioactivity, the atomic bomb, and the use of isotopes. The author includes five chapters on civil defense organization, with its medical and special health services. The last chapter deals briefly with radiological defense. The general practitioner should be interested in Section II with its discussion of the symptomatology, pathology, and treatment of atomic bomb injuries and radiation sickness. It is questionable, however, whether the general practitioner is interested or has the background to read and understand atomic theory with nuclear fission and chain reaction.

It is not a specific book for veterinarians. Section II might be of interest to the small animal practitioner and to those who are seriously interested in atomic warfare and nuclear energy.—[*The Physician in Atomic Defense. By Thad P. Sears. 290 pages. 53 illustrations. The Year Book Publishers, Inc., Chicago, Ill. 1953. Price $6.00.*]—F. A. TODD.

Genetics of the Dog

The author introduces "The Genetics of the Dog" with a presentation of the mendelian theory. A number of definitions of fundamental terms are given making it unnecessary, yet desirable, to have a prior knowledge of genetics. Reproduction in the dog is adequately presented, and the hereditary factors pertaining to the subject are discussed. The conformation, type, and size of breeds, together with the influence of hereditary factors, is carefully summarized. In the chapter on physiological peculiarities, the author cites 38 hereditary diseases and defects, together with the mode of inheritance and references for each.

A full chapter is devoted to hereditary influences on behaviors and mental attributes. The hereditary influences on the abilities, interest, and willingness of an animal to do the particular work for which he is bred, is discussed. Twenty pages are devoted to a discussion of hereditary influences on the coat, skin, and color, and six more pages to the use of the genetic formulas in color breeding. The inheritance of pigmentation appears to be complex. The expected results from color crosses are given in a chart.

In the final chapter, devoted to theory and practice in dog breeding, the author indicates that the key to success is the proper combination of science and practical dog breeding. The author recommends that for any period of time a few desirable traits in a breed be actively selected and a few nondesirable ones be carefully avoided; that a scoring system be developed to evaluate the results; and that line breeding be used to strengthen the strain.

The author ably handles the subject of genetics in the dog. The appendix greatly aids the nontechnical reader by defining terms and suggesting record forms. A 16-page list of references is of value to the reader interested in canine genetics. The index is adequate.

The book should be informative to the dog fancier, and interesting as well as enlightening to the veterinarian who deals with small animals. —[*The Genetics of the Dog. By Marca Burns. 96 pages. Illustrated. Commonwealth Agricultural Bureaux, Farnham Royal, Slough, Bucks, England. 1952. Price $1.75.*]—G. W. MATHER.

Textbook of Meat Inspection

This textbook is intended for students and as a reference book for those engaged in meat inspection and other food control work. In addition to discussing meat inspection, the inspection of rabbits, poultry, and fish is also included as well as a revision of the sections of Dr. Thornton's first book which dealt with bacteriological examination of meat as an aid in making dispositions. The chapter on comparative anatomy of the tissues and organs includes a comprehensive coverage of the location and function of the lymph nodes and organs of significance in meat inspection.

The textbook appears to be written particularly for inspectors in a country with a cool climate, with limited facilities for refrigeration, and one which imports a large part of its meat supply.

A great deal of emphasis is placed on justifying a change in viewpoint and methods which would make available for food, meat from animals affected with conditions which under most inspection systems is required to be condemned. The author adequately treats those diseases and conditions known to be transmissible to man, or which might cause food poisoning, but leaves the reader convinced that inspection must give the consumer assurance that his meat came from healthy animals and was produced under sanitary conditions. The suggestion that "boiling and cooking are sufficient to destroy any smear of tuberculous material on the surface of a joint of meat" does not give proper emphasis to training workmen and inspectors to prevent such serious contamination.

Environmental sanitation, plant construction and lay-out, equipment, and methods of operation described are not up to present day standards in several other countries. Many of the methods described are regarded as antiquated by the American meat packer. While the author briefly mentions welfare facilities for workmen, he does not associate the need for such facilities with good sanitation. Recommendations for providing separate "guttery and tripery" facilities away from slaughtering facilities for preparing edible tripe, edible fat and casings, is contrary to the best practices in this field which require sanitary handling of these products.

In describing food poisoning, the author shows that the practice in some countries of selling uninspected meat of questionable wholesomeness is not new.

The discussion concerning the acceptability of

animals which have died other than by slaughter leaves one with the impression that some of these animals might be passed for food. The author's treatment of the practice of slaughtering diseased animals and presenting them dead and unevisceratedfor inspection is not in accordance with recognized practices in many other countries.

In his chapter on bacteriology of meat, the author brings together much valuable information involved in conditions affecting the wholesomeness of meat.

The last chapter, covering the inspection of fish, furnishes a good description of the distinguishing features of marketable edible fish, shellfish, and molluscs. An interesting procedure for purification of oysters and molluscs is described.

The book is well illustrated and will serve as a valuable reference book. It is, however, not recommended for use as a textbook in the United States because it does not recognize consumer expectancy as it exists in this country, and the terms used and the problems and practices described are also different from those in the United States.—*[Textbook of Meat Inspection. By Horace Thornton. 619 pages. Illustrated. 2nd ed. Printed in Great Britain; available from Alexander Eger, Inc., Chicago. 1952. Price $10.50.]*—C. H. PALS.

Stedman's Medical Dictionary

The new revised edition of Stedman's Medical Dictionary contains several thousand additional words and 600 more illustrations than the previous edition.

We are in full accord with the suggestion made in the Preface by the editors that some philological authority (an association or academy) be established which could approve or disapprove proposed new words in medical literature. The need for new words is a healthy sign, we agree, but too often little thought is given to the forming of those words by the introducer. The editors say ". . . terms have been produced which scarcely deserve to be placed in any category more dignified than that of jargon." Yet they conclude that no matter how much an editor of a medical dictionary may dislike these ill-formed words, he can not be too much of a purist and must not, except in extreme instances of bad word-forming, deny them space. This would seem to uphold our contention that because a word is in the dictionary, it is not necessarily correct.

Attention has been given to the addition of more veterinary medical terms. As an example, we were pleased to find Teschen's disease listed and defined. It does not appear in two other widely used medical dictionaries we have consulted. However, scrapie, a disease of sheep is incorrectly defined, as in other medical dictionaries. Our criticism of the medical dictionaries we have used is that they are weak in veterinary nomenclature. But each edition of most of them contains more than the former, which is encouraging. Perhaps in the next edition the editors of Stedman's may be able to include a really comprehensive list of veterinary medical terms.—*[Stedman's Medical Dictionary. Edited by Dr. Norman B. Taylor, University of Western Ontario, and Lt. Col. Allen Taylor. 18th ed. 1,561 pages. Illustrated. The Williams & Wilkins Co., Baltimore, Md. 1953.]*—H.S.B.

Nutritional Physiology of Domestic Animals

This publication, dealing with the nutritional physiology of domestic animals, is divided into five sections. Each section has a general introduction to the physiological processes of pregnancy, growth, lactation and work, considering energy, protein, and other nutritional requirements.

There are 160 pages, 37 illustrations, 92 tables, and a number of references in the text and at the end of each chapter.

This little book is a worth-while contribution to the literature of animal nutrition.—*[The Introduction into the Nutritional Physiology of the Domestic Animals. (Einfuhrung in die Ernahrungsphysiologie der Haustiere). By Walter Lenkeit. Ferdinand Enke, Stuttgart. 1953. Price 26 marks.]*—F. KRAL.

REVIEWS OF VETERINARY MEDICAL FILMS

This Little Pig.—Sound, color, 16 mm., running time about thirteen minutes; produced and procurable from Commercial Solvents Corporation, Terre Haute, Ind.

This film traces the search for a method of supplying antibiotics to baby pigs early in life before antibiotic creep feed is consumed, and during a critical period when heavy death losses are sustained. Various unsuccessful attempts are illustrated and explained and the method, developed by Commercial Solvents Corporation, of subcutaneous implantation of bacitracin pellets is dealt with in detail.

Investigative and production procedures for bacitracin pellets are shown and results of controlled and field experiments are given indicating livability, disease prophylaxis, and litter uniformity. The picture includes both direct photography and animated cartooning. It is well produced and should be of interest to professional audiences.

Leading a Dog's Life.—The National Dog Welfare Council estimates American dogs at 22,000,000, owned by 17,000,000 American families. Whereas twenty-five years ago, the average dog ate only left-overs from the table, today Americans spend $175,000,000 a year on prepared dog foods. Another $5,000,000 is spent for collars, leashes, blankets, nail clippers, cushions, toys, et cetera.—*Atlanta J., April 6, 1953.*

THE NEWS

Report on Fifteenth International Veterinary Congress in Stockholm

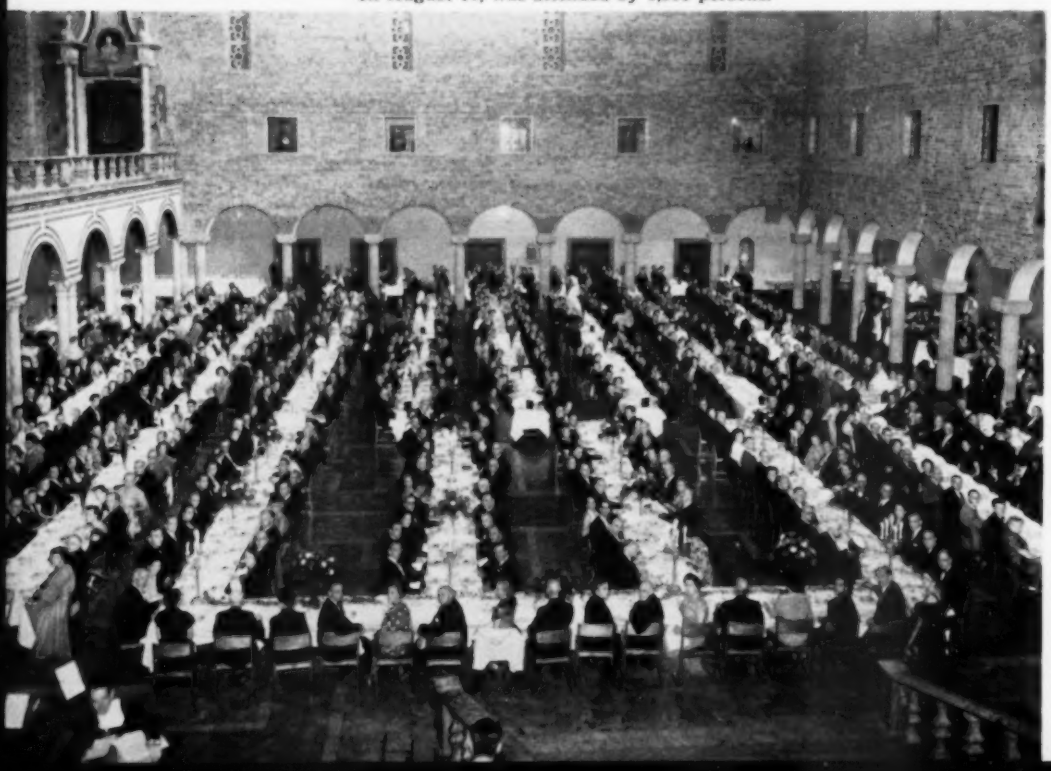
On Saturday, Aug. 15, 1953, the closing session of the Fifteenth International Veterinary Congress was held in the Concert Hall of Stockholm, Sweden, concluding a solid week of plenary sessions, section meetings, official functions, visits to places of interest in and around the city, and entertainment events, all of which were exceptionally well planned, arranged, and carried out by the Organizing Committee of the Congress.

Stockholm was a delightful setting for the Congress, the facilities were good, and the nearly 1,500 delegates and wives enjoyed the fine weather and the many delightful attractions which this "Venice of the North" affords. Delegations were present from some 56 countries, the largest, outside of the Scandinavian countries, being those from England, France, Germany, and the United States.

The first event of the Congress week was the unofficial or informal reception on Sunday evening August 9 in the beautiful "Blue Room" of the Town Hall and attended by upward of a thousand persons as guests of the Congress. The opening session convened in the large theater of the Concert Hall on Monday morning, August 10, where the address of welcome was given by Dr. Axel Alegren, chairman of the Organizing Committee, followed by an opening address by the Honorable Sam Norup, Minister of Agriculture. The following officers of the Fifteenth Congress were then elected: president, Dr. Karl Ericksson, Sweden; vice-presidents, Dr. W. A. Hagan, U. S. A., Dr. C. Bressou, France, and Dr. A. Hjärre, Sweden; secretary general; Dr. Axel Isaksson, Sweden; treasurer, Dr. G. T. Kranz, Sweden.

The following honorary officers were elected: honorary presidents, Dr. B. T. Simms, U. S. A.; Dr. L. deBlieck, Netherlands; Sir Daniel Cabot, England; Dr. J. Fortner, Germany (west zone); Dr. F. Meissemeier, Germany (east zone); Dr. E. Ramon, France; Dr. G. Sanz Egana, Spain;

The Fifteenth International banquet held in the Blue Room of the Town Hall, Stockholm, on August 11, was attended by 1,200 persons.



Dr. K. I. Scriabine, U.S.S.R.; honorary vice-presidents, Dr. A. Alegren, Sweden; Dr. G. Forsell, Sweden; Dr. H. Magnusson, Sweden; and Dr. Sven Wall, Sweden.

Following President Ericksson's address, speeches were made by Sir Daniel Cabot, president of the Permanent Committee of the I. V. C.; Dr. I. Altara, of the International Office of Epizoötics; Dr. R. V. L. Kesteven, FAO (United Nations); Dr. M. M. Kaplan, WHO; Dr. S. Koch, of the International Dairy Federation; and Dr. B. L. Rowan, Office of European Economic Coöperation.

Plenary Lectures

The first plenary lecture of the Congress was delivered by Sir Thomas Dalling of Great Britain, a most scholarly dissertation on "The Veterinary Profession — Past, Present, and Future." In addition to the lecture by Sir Thomas Dalling at the opening session, lectures were also given at plenary sessions throughout the week by:

Professor G. Flückiger, Switzerland — Efforts Realized to Date in the International Control of Epizoötics.

Professor F. Schönberg, Germany — The Place of the Veterinarian in Food Sanitation.

Dr. James H. Steele, U. S. A. — Relation of Milk Sanitation to Communicable Disease Control and Public Health.

Professor T. Seekles, Netherlands — The Biochemical Approach to Animal Diseases.

Professor H. Simmonet, France — Hormonal Regulation of Metabolism.

Professor T. Bonnadonna, Italy — Artificial Insemination and the Veterinary Profession.

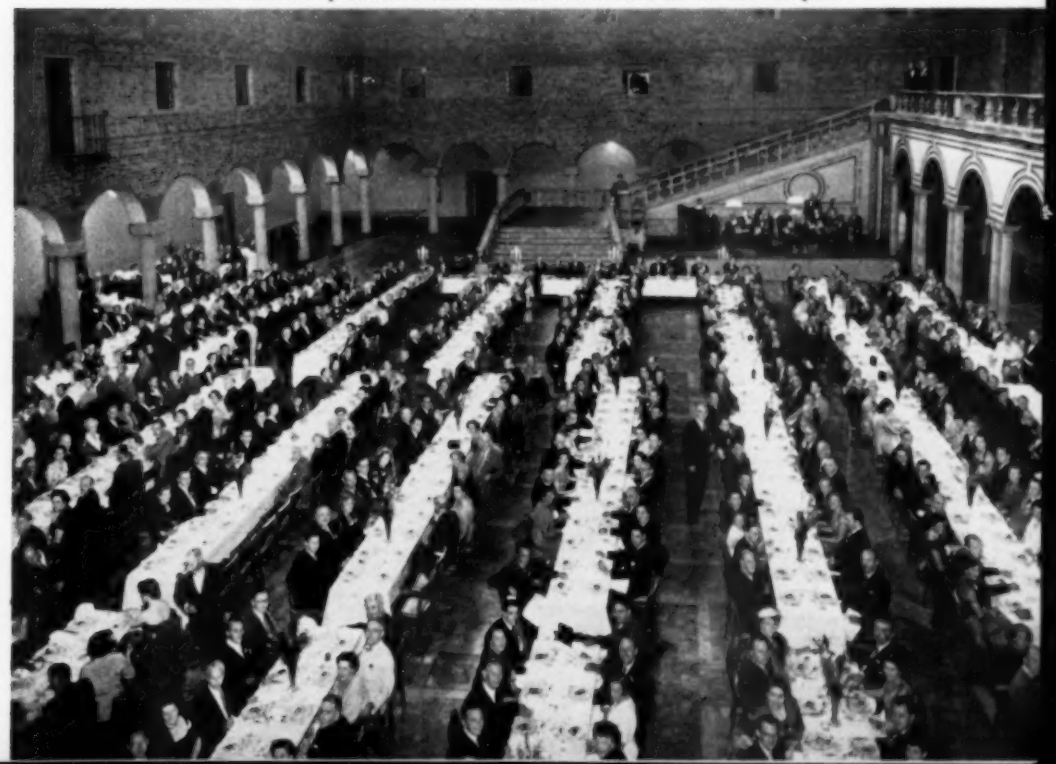
Dr. M. G. Fincher, U. S. A. — Etiology, Prevention and Treatment of Bovine Mastitis.

Professor J. Dobberstein, Germany (eastern zone) — The Work of the Veterinarian in Food Hygiene, with Special Reference to Foods of Animal Origin.

Section Meetings

Most of the more than 260 papers at the Congress were presented in the eight sections into which the program was divided and which met in the afternoons. Space does not permit complete listing of the section papers, so that only those reports by authors from the United States will be given here. The Organizing Committee had done a splendid job of obtaining in advance, editing and publishing—in two volumes comprising some 1,200 pages—most of the section papers so that Congress delegates received copies at the time they registered, thus having access at once to the complete papers, or summaries of them, that were to be presented. The authors were then given about ten minutes each to recapitulate the main points of their reports or to demonstrate the points with lantern slides or films.

Picture of the Congress banquet taken from the opposite end of the room and showing the staircase used by some 90 waiters in formation who served the banquet.



Discussion of papers in any given field then followed.

The "Proceedings of the Fifteenth I. V. C.", Part I, is in two volumes and contains the 261 section papers. Part II will contain the plenary lectures, other speeches, discussions of section papers, and bibliographies of same; also resolutions adopted by the Congress and other data. Orders for the complete Proceedings may still be sent *direct* to: Secretariat, XVth International Veterinary Congress, c/o Dr. Axel Isaksson, Stockholm 50, Sweden. The price is 45 Swedish kroner ($9 U. S.) postpaid, and remittance should accompany order.

SECTION PAPERS BY U. S. VETERINARIANS

As a result of nominations made by the U. S. Committee some time ago, the Organizing Committee of the Congress selected a considerable number of topics to be presented by United States veterinarians at sectional meetings. The authors and their subjects were:

Section IA—Bacterial Diseases

H. S. Cameron — Comparative Metabolic Activity of the Genus Brucella.

A. G. Karlson (with W. H. Feldman) — Mycobacteria of Human Origin Resembling *Mycobacterium Avium*.

Section IB—Viral Diseases

Ervin Eichhorn — Laboratory Diagnosis of Foot-and-Mouth Disease.

C. A. Brandly — Epizoötiology and Spread of Newcastle Disease.

Ernest S. Tierkel (with M. M. Kaplan) — Advances in the Control of Rabies.

Herbert S. Stoenner — Observations on the Epizoötiology of Bovine Q Fever.

K. F. Meyer — Distribution and Identification of Psittacosis Viral Agents in the Animal Kingdom.

R. E. Kissling — Host Vector Relationships in Eastern Equine Encephalomyelitis.

Section II—Parasitic Diseases

C. R. Cole, *et al.* — Toxoplasmosis in Domestic Animals.

Section III—Poisonings

L. K. Bustad — Toxicity of Radioiodine.

Section IV—Physiology, Pathological Physiology, Deficiency Disease

Colonel John H. Rust — Total Body Irradiation of Various Species of Animals — Some Physiologic and Pathologic Effects Evident in Blood, Bones, and Thyroid Gland.

Section V—Physiology and Pathology of Reproduction and Lactation (no speakers from the U. S. in this section.)

Section VI—Animal Husbandry

George H. Hart — Nutrition, Disease, Livestock Well-Being, and Human Welfare.

Section VII—Diagnosis, Therapy, and Surgery

Jacques Jenny — Motion Picture Review of Modern Fracture Treatment.

Gerry B. Schnelle — Radiographic Diagnosis of the Abdominal Mass.

Erwin Jungherr — Neuropathologic Differentiation of Symptomatic Paralysis in Fowl.

A. D. Goldhaft — A Registry of Bird Tumors.

David Hopkins — Private Veterinary Practice in America.

S. F. Scheidy — Recent Advances in Chemical and Antibiotic Therapy.

Section VIII—Food Hygiene and Public Health

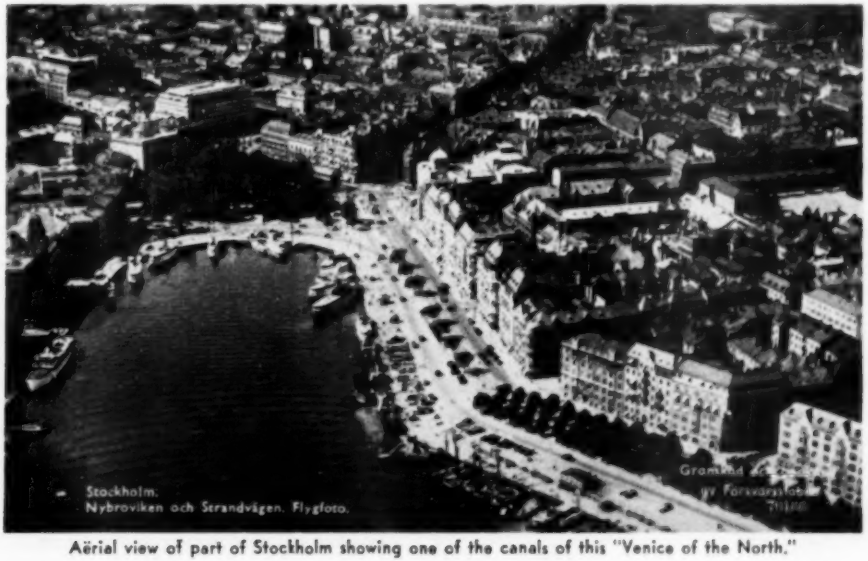
M. M. Kaplan — The Concept of Veterinary Public Health and Its Application in the World Health Organization.

Scene at Ulvik in the mountain and fjord country of northern Norway, one of the scenic spots visited by the tour party from the States.

Night-time picture of the "Stadshuset" or Town Hall of Stockholm, in which social functions of the Fifteenth International Veterinary Congress were held.

Robert Rausch — Animal-Borne Diseases in Alaska and their Public Health Significance.

The following persons from the United States also served as section officers during the Congress: Drs. K. F. Meyer, chairman, Section on Bacterial Diseases; Erwin Jungherr, vice-chairman, Section on Diagnosis, Therapy, Surgery and Patho-anatomy; C. A. Brandly, chairman, Section on Viral Diseases; and L. K. Bustad, vice-chairman, Section on Poisonings.



Aërial view of part of Stockholm showing one of the canals of this "Venice of the North."

VISITS TO PLACES OF VETERINARY INTEREST

The Organizing Committee had arranged a series of visits and tours to places of interest both in Stockholm and outside the city, so that delegates and others were able to select those in which they were interested and visit them during the afternoons of the Congress week. Outstanding among these were: the Royal Veterinary College, the State Veterinary Medical Institute, the National Institute of Public Health, the State Bacteriological Laboratories, the Entkoping A. I. Association (laboratory and bull station), the Royal Agricultural College and the National Experiment Station (in Uppsala), the Wiad Animal Breeding Institute; also several outstanding as well as representative farms.

Conducted postcongress tours were also offered to places of special interest in South Sweden and West Sweden.

AVMA CONGRESS TOURS SUCCESSFUL AND WELL-CONDUCTED

The total delegation from the United States at the Congress numbered more than 100 persons, including veterinarians and their wives or other family members. Of the total, about 60 persons comprised the main tour party which left Toronto on the night of July 23 following the close of the AVMA convention and traveled by boat train to Montreal, sailing the next day on Canadian Pacific's *Empress of France*. After arrival in Liverpool, about one week was spent in Scotland and Norway before arrival in Stockholm for the Congress.

After the Congress, the main tour party visited points of interest in Denmark, Holland, Belgium, France, Germany, and Switzerland before returning to England; others went to Italy before returning to England prior to departure for the states. The tours, which were arranged by the AVMA in collaboration with Travel Service Bureau of Brookline, Mass., were well managed and conducted throughout.

Final Report on 1952-1953 Research Fund Campaign

Contributions to the one-year campaign, which began June 23, 1953, to raise funds for fundamental veterinary research totaled $39,242.39, as of Sept. 17, 1953.

Donations from individual veterinarians amounted to $26,825.39, an average of $12.42 from each of the 2,159 veterinarians who contributed to the fund during the campaign.

In addition to individual contributions received, state, provincial, and local associations have donated a total of $5,285.00; women's auxiliaries have given $1,192.00; and commercial companies and other groups have contributed $5,940.00.

Members also are urged to read the annual report of the Research Fund-Raising Committee which will appear in the 1953 "Proceedings Book" of the Ninetieth Annual Meeting of the AVMA.

Fifth and Final Report on 1952-1953 AVMA Research Fund Campaign Prepared Sept. 17, 1953

State	Total veterinarians	Contributions (to date) (No.)	(Amount)
Alabama	226	23	$ 285.00
Arizona	71	15	180.00
Arkansas	69	14	87.00
California	1,332	137	1,648.00
Colorado	210	35	455.00
Connecticut	138	35	671.00
Delaware	42	11	147.00
District of Columbia	73	42	405.00
Florida	300	30	540.00
Georgia	280	21	272.00
Idaho	88	12	98.00
Illinois	800	156	2,374.00
Indiana	580	72	868.00
Iowa	814	115	1,335.00
Kansas	375	49	540.00
Kentucky	245	21	304.50
Louisiana	145	11	110.00
Maine	73	11	92.00
Maryland	149	31	442.00
Massachusetts	300	24	305.00
Michigan	623	87	1,007.06
Minnesota	525	70	778.00
Mississippi	128	16	202.50
Missouri	460	58	644.00
Montana	93	8	80.00
Nebraska	330	42	470.00
Nevada	30	2	15.00
New Hampshire	55	5	40.00
New Jersey	346	63	856.50
New Mexico	46	11	105.00
New York	1,161	144	2,258.00
North Carolina	207	35	460.00
North Dakota	70	9	115.00
Ohio	940	118	1,173.00
Oklahoma	220	25	493.50
Oregon	198	22	255.00
Pennsylvania	640	100	1,227.50
Rhode Island	28	2	50.00
South Carolina	110	5	30.00
South Dakota	119	14	181.00
Tennessee	168	21	517.50
Texas	875	135	1,078.45
Utah	68	16	252.00
Vermont	74	11	107.50
Virginia	180	38	454.50
Washington	314	49	590.00
West Virginia	78	6	90.00
Wisconsin	664	112	1,354.00
Wyoming	55	7	80.00
Hawaii	21	6	95.00
Canada	—	55	611.88
Canal Zone	—	1	5.00
Foreign	—	1	10.00
Total	15,136	2,159	$26,825.39

Other Contributions:		
Veterinary Associations:	$5,285.00	
Women's Auxiliaries:	$1,192.00	
Commercial Companies etc.	$5,940.00	$12,417.00
		$39,242.39

News From Washington

Special Registrants.—Selective Service System issued instructions on September 22 requesting local boards to discontinue physical examination and induction of **special** registrants for the Armed Forces for the remainder of the fiscal year.

This action was based on Selective Service being advised the Armed Forces **do not expect** to levy a call for physicians or veterinarians at least prior to July 1, 1954.

Dual Status Registrants.—Of special interest to veterinarians who are **regular** draft registrants under Section 3, Universal Military Training and Service Act (therefore subject to **induction as privates),** is the announcement, Oct. 2, 1953, by the Selective Service System, to state directors, advising them to **postpone induction** of all regular registrants who are also special registrants, except delinquents and volunteers, pending revision of Operations Bulletin 88.

It is understood this action was taken to conserve physicians, dentists, and veterinarians until such time as they will be needed by the Department of Defense as commissioned officers in a professional capacity. To effect this, it is understood, all physicians, dentists, and veterinarians who qualify will be afforded an opportunity to accept appointment in the Reserve Corps as officers. They will not be ordered into military service **involuntarily** until needed. Should such appointment be **declined,** the individual **will be subject to induction** by local draft boards, as his name will remain in the Selective Service files as a regular draft registrant under Section 3, Universal Military Training and Service Act.

If and when this policy goes into effect, individuals affected will be advised by their local draft boards relative to procedure, etc.

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Defense Department Directive.—The Department of Defense, on October 7, issued a directive to implement the revised doctor draft law (P.L. 84, 83rd Congress); thus, it is prepared to put into effect the changes provided in the new law. An important new point is the upward adjustment in grade affecting certain officers. Public Law 84 requires the appointment, re-appointment, or promotion to a grade "as may be commensurate with his professional education, experience, or ability." The directive, while retaining the present grade scale based on experience, states promotion must be given to officers now on active duty who qualify for a higher grade.

In effect, this means professional experience alone will determine the grade in which a veterinarian serves. It eliminates such limiting factors as shortage of higher grades in tables of organization, the commissioning of those in priorities I, II, and III on the basis of cut-off dates, and delays pending action of officers' boards.

The following scale is used to determine the grade for veterinarians: less than one year professional experience since graduation, second lieutenant; one year but less than five, first lieutenant; five years but less than twelve, captain; twelve but less than nineteen, major; nineteen or more,* lieutenant colonel.

*Veterinarians, with twenty-six or more years of professional experience, who have achieved national prominence as authorities in their particular specialty, may be appointed in the grade of colonel.

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Mr. Walter Thurston, former ambassador to Mexico, has been appointed **director of the U. S. Section of the Joint Mexican-U.S. Commission** in charge of the current foot-and-mouth eradication campaign. Mr. Thurston was ambassador to Mexico during the previous eradication campaign.

Washington office address—Brig. General J. A. McCallam, Room 109, 1507 M St., N. W., Washington 5, D. C.

WOMEN'S AUXILIARY

The Beginning of the Women's Auxiliary to the AVMA.—The Women's Auxiliary to the AVMA was born in the throes of war. It was only natural that the wives who had accompanied their husbands to the AVMA meeting at Kansas City, Aug. 20, 1917, should wish to band together to help their country.

A meeting was planned and all the women were invited to attend. At that time, we formed the Ladies' Auxiliary to the AVMA. I would like to quote from the September, 1917, issue of the *American Journal of Veterinary Medicine*:

> A step which may prove a very important one was taken at this meeting—the organization of a ladies' auxiliary to the AVMA. The destinies of the new organization were entrusted to the following officers during the coming year: Mrs. W. Horace Hoskins, president; Mrs. A. T. Kinsley, first vice-president; Mrs. R. P. Marsteller, second vice-president; Mrs. T. H. Ferguson, third vice-president; Mrs. Charles E. Cotton, recording secretary; Mrs. Ashe Lockhart, corresponding secretary; Mrs. H. Jensen, treasurer.
>
> The organization begins with a membership of 55. It is expected that it will be permanent and find many opportunities for forwarding veterinary interests and veterinary service in the country. Just at this time it is giving consideration primarily to what it may do to aid in the war. It is expected that such aid and comfort as the members may be able to give will be tendered to the families of veterinarians who engage in military duties that take them from their homes. They also expect to lend their aid to the AVMA in raising a fund for the relief of veterinarians of the allied armies (including, of course, the United States) and their families.

And even then the American way of combining business and pleasure was in vogue, for the following item was also in the JOURNAL:

> The ladies, who accompanied the veterinarians to Kansas City, carried away memories of one of the pleasantest weeks they had ever enjoyed. The ladies of the local committee had planned for their entertainment during every day and almost every waking hour. Among the social features might particularly be mentioned auto tours, shopping tours, a card party at the Hotel Muehlbach, a visit to Electric Park, a visit to Armour's packing plant, a visit to the National Biscuit Company, a banquet, luncheons, etc.

The early years of our organization were indeed rugged. Because many of the women had not yet formed the habit of accompanying their husbands to the meetings, it was difficult to get officers. They, therefore, asked the president to serve two years and the vice-presidents were chosen from the locations in which the AVMA annual meeting was to be held so they might preside if the president could not attend.

The early secretaries really had the difficult task, as Mrs. Marsteller or Mrs. S. E. Hershey could tell you, for they might write to five or six women before finding one who would attend and respond to the address of welcome, or take some other part of the program. Many times Mrs. Hershey has written to me two or three days before the meeting or on the first day of the meeting would say, "Mrs. —— isn't coming" or "I didn't hear from Mrs. ——, will you respond to the address of welcome?" or "Will you be chairman of the nominating committee?" She knew I would be there if possible. (I've only missed four meetings since I started attending in 1916.)

It was soon found that our money was not needed essentially for needy veterinarians, so in July, 1922, the Auxiliary offered to lend a sum not to exceed $350 to a senior attending a recognized veterinary college in the United States or Canada, that amount to receive interest at 4 per cent and to be refunded to the Auxiliary in two yearly payments; and in a report which Mrs. Marsteller made in August, 1924, $875 was loaned to students and $25 had been spent for relief from 1920 to 1924. So you see, we were building our structure upon a firm foundation, and I have watched it grow from an organization composed of a few earnest women with the purpose in mind of assisting the families of veterinarians who were in World War I, to a flourishing auxiliary of more than 3,000 women interested in the welfare of our veterinary students and in informing the public of the value of veterinary service.

It has withstood the test of time, from the days when we traveled by slow train or in cars that had only side curtains to keep out the rain or sand, to the present when we travel to any of our cities in luxurious cars, trains, or planes; and I am proud to belong to an auxiliary which seeks to serve, as well as to enjoy the pleasure of friendly association.

s/MRS. JAMES D. GROSSMAN, *Columbus, Ohio.*

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Central California Auxiliary.—On August 25, the Women's Auxiliary to the Central California Veterinary Medical Association met at the home of Mrs. C. T. Lambert in Visalia. The guest speaker was Mrs Alberta Houk of the State Department of Education, who discussed the orientation and teaching of the blind.

Fifteen members attended the July meeting of the Auxiliary which was held in the home of Mrs. Helen LaRue in Fresno.

s/(MRS. I. C.) VIRGINIA MCDONALD, *Secretary.*

APPLICATIONS

Applicants — Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative By-Laws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent association shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. This certification was made by the secretary of the constituent association in accordance with Section 2, Article X, of the Administrative By-Laws.

First Listing

CARAWAY, CHARLES T.
3401 Orleans Dr., Nashville, Tenn.
D.V.M., Texas A. & M. College, 1946.

CRAIG, EUGENE L.
1843 N. Gettysburg Ave., Dayton, Ohio.
D.V.M., Ohio State College, 1950.

GRAHAM, KENNETH D.
P. O. Box 101, Claresholm, Alta.
D.V.M., Ontario Veterinary College, 1952.

GRIFFIN, WILLIAM B.
1415 W. 3rd St., Elk City, Okla.
D.V.M., Texas A. & M. College, 1945.

METHOT, ARMAND
Cushing St., Acton-Vale, Que.
D.V.M., St. Hyacinthe Veterinary School, 1949.

PECK, GEORGE K.
R. R. 1, Oakville, Ont.
D.V.M., Ontario Veterinary College, 1950.

RISTIC, MIODRAG
1105 Ridgwood Ave., Madison, Wis.
D.V.M., Hannover Veterinary College, 1950.

SHAW, LAGRANDE C.
331 No. 11th St., Geneva, Neb.
D.V.M., Kansas State College, 1938.

ZWEIG, HARRY M.
Nassau, N. Y.
D.V.M., Ohio State University, 1938.

Applicants — Not Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative By-Laws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and the names of his endorsers.

First Listing

BERGER, JOSEPH H.
Office of Air Surgeon, Hq. 12th Air Force, APO 12, c/o PM, New York, N. Y.
D.V.M., Ohio State University, 1939.
Vouchers: W. O. Kester and J. B. Couch.

CLAYTON, FREDERICK W.
10004 Crestwood Rd., Kensington, Md.
D.V.M., Ohio State University, 1944.
Vouchers: J. H. Hempy and J. B. Couch.

FIGUEROA, JUAN
Apartado 23, Barranco, Lima, Peru.
D.V.M., Ohio State University, 1944.
Vouchers: J. G. Hardenbergh and H. E. Kingman, Jr.

RILEY, WILLIAM E.
Army and Navy Hospital, Hot Springs, Ark.
D.V.M., Texas A. & M. College, 1944.
Vouchers: H. R. Ellis and D. W. Hubbard.

Second Listing

LYDAY, CHARLES V., 167th Medical Detachment, APO 154, U. S. Army, New York, N. Y.

1953 Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative By-Laws to members in good standing of student chapters. Applications from this year's senior classes not received in time for listing this month will appear in later issues. An asterisk (*) after the name of a school indicates that all of this year's graduates have made application for membership.

First Listing

Alabama Polytechnic Institute*

BAKER, OSBORNE E., JR., D.V.M.
Box 113, Nichols, S. Car.
Vouchers: R. S. Sugg and W. S. Bailey.

BRANSON, JOSEPH W., D.V.M.
9 McDonough Blvd., S. E., Atlanta, Ga.
Vouchers: C. S. Roberts and R. S. Sugg.

Colorado A. & M. College

KEYSER, RAYMOND C., D.V.M.
Box 546, Ronan, Mont.
Vouchers: L. K. Wayt and R. H. Jourdan.

Iowa State College

BOWNE, JOHN G., D.V.M.
437 Pammel Court, Ames, Iowa.
Vouchers: R. Getty and I. A. Merchant.

Michigan State College

NEWBURY, FRED L., D.V.M.
2823 S. Harlem Ave., Berwyn, Ill.
Vouchers: L. E. Fisher and M. H. Gooch.

University of Pennsylvania

LEHMAN, ROBERT J., V.M.D.
1611 Barringer St., Philadelphia, Pa.
Vouchers: H. E. Schaden and D. G. Lee.

MARX, ELMER E., JR., V.M.D.
192 E. High St., Waynesburg, Pa.
Vouchers: P. C. Snoke and C. H. Beckman.

McCLURE, JOHN P., JR., V.M.D.
Box 471, Tonasket, Wash.
Vouchers: R. S. Nelson and H. D. White.

Texas A. & M. College

PIGOTT, MARTIN B., JR., D.V.M.
326 Claremore, Corpus Christi, Texas.
Vouchers: J. M. Prewitt and J. M. Spriegel.

Second Listing

University of California*

JOHNSON, LOUIS F., D.V.M., c/o Brighton Animal Hospital, Rt. 2, Box 3810, Sacramento, Calif.

Colorado A. & M. College

CLARK, SIDNEY H., D.V.M., Molina, Colo.
MAY, HARRY A., D.V.M., Rt. 3, Plant City, Fla.
MURSKO, FRED P., D.V.M., 775 N. Oregon St., Ontario, Ore.

University of Illinois*

KINGRY, EARL L., D.V.M., Seaton, Ill.

Iowa State College

GILLETTE, D. DALE, D.V.M., Black Hills Veterinary Supply, Hermosa Rd., Rapid City, S. Dak.

University of Michigan

RILETT, WALLACE J., D.V.M., 110 N. Milwaukee, Waterford, Wis.
SIEGEMAN, JAMES H., D.V.M., 780 Forest, Birmingham, Mich.

University of Missouri

WHEATLEY, HILERY E., D.V.M., 340 Gladstone, Jacksonville, Ill.

University of Pennsylvania

GARLICK, ELTON C., V.M.D., Box 883, Kannapolis, N. Car.
HATHAWAY, E. JEFFERIES, V.M.D., Clifton Park Manor, Apt. 73-5, Wilmington, Del.
NEUFER, FRANKLIN E., V.M.D., 307 S. Broad St., Kennett Square, Pa.
SEVOIAN, MARTIN, V.M.D., New York State Veterinary College, Cornell University, Ithaca, N.Y.

Texas A. & M. College

ANDERSON, WILLIAM L., D.V.M., 5705 E. Belknap, Ft. Worth, Texas.
BENSON, THOMAS L., D.V.M., 149 Arlington St., San Angelo, Texas.
CROW, EMIL W., D.V.M., 53 Bliss St., Springfield, Mass.
DAVIS, JOHN P., JR., D.V.M., 156 Taliaferro, Shreveport, La.
GOODMAN, JOHN G., D.V.M., 3300 S. Congress Ave., Austin, Texas.
HOWARD THADDEUS M., JR., D.V.M., 1918 W. Woodlawn Ave., San Antonio, Texas.
MADDOX, REX M., D.V.M., Rt. 3, Alvarado, Texas.
PHILLIPS, EULA JR., D.V.M., 2300 N. Main St., Victoria, Texas.
PRICE, JACK W., D.V.M., 3301 McCullough Circle, Houston, Texas.

COMMENCEMENTS

Alabama Polytechnic Institute.—At the 1953 commencement exercises of the Alabama Polytechnic Institute, 60 candidates were presented for the D.V.M. degree.

H. S. Adair, Jr.
R. P. Akers
W. J. Austin
O. E. Baker, Jr.
R. L. Baldwin
R. E. Becker
R. A. Bennett, Jr.
Michael Beven
J. W. Branson
C. M. Byles
R. M. Byrd
L. C. Cardinal
J. E. Chancellor
C. G. Cline
J. C. Cooksey
J. C. Cornwell
W. S. Fairey
E. J. Fievet
L. Y. Frost
C. F. Gaines
C. F. Giddens
R. A. Glass
B. I. Grissett
J. A. Gunn
H. H. Hayes
H. E. Henderson
T. M. Higginbotham
C. B. Hill
J. H. Hunt
J. G. Martin
W. L. Martin
I. J. Mitchell
J. P. Morgan
P. J. Muller
R. S. Mullins
C. C. Murphy
J. B. Murphy
J. H. Nelson
E. E. Nissen
J. W. Patterson
J. B. Pettway
B. G. Pratt
J. R. Priester
A. J. Rimes
S. B. Seltzer
E. C. Sharman
G. P. Shiflett
F. L. Shuler
C. E. Smith, Jr.
J. H. Stevenson
J. L. Stookey
A. F. Straughn
H. A. Tennant
Jo N. Thomas
J. W. White
B. L. Whitt
H. S. Wilcox
R. E. Wilhite
G. F. Yopp
J. C. Young

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University of California.—At the commencement exercises of the University of California on June 17, 1953, 52 candidates were presented for the D.V.M. degree.

R. B. Abbott
J. P. Barnier
E. V. Bayer
J. W. Bayliss
J. L. Bittle
J. S. Blackard
L. H. Brazil
J. F. Chapman
W. Q. Chin
H. G. Clute
K. G. Cooke
T. B. Condon
M. N. Corfl
C. E. Cornelius
A. A. Cuthbertson
L. H. Darrow
J. R. Davies
Evelyn Dean
O. J. Eggers
A. J. Eisenhower
K. E. Erwin
R. Y. Foos
J. C. Hanson
R. E. Harris
T. A. Holliday
R. L. Hosker
R. A. Jackson
L. F. Johnson
R. A. Kathein
W. M. Kortum
R. F. Larson
B. V. Lundberg
R. K. Mortenson
W. K. Newshutz
A. D. Newell
G. D. Pettit, Jr.
J. P. Pilock
A. C. Pier
W. J. Pimentel
L. D. Price
S. E. Rood
M. J. Silver
R. F. Simpson
J. H. Steere
L. R. Tangney
G. S. Troxell
J. A. Tucker
D. L. Van Pelt
W. M. Vansell
W. L. Vowles
G. W. Wallis
W. W. Watkins

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Colorado A. & M. College.—At the 1953 commencement exercises of the Colorado A. & M. College, 59 candidates were presented for the D.V.M. degree.

R. G. Anderson
F. W. Arnold
R. A. Bagley
J. R. Barton

T. H. Belling
Harold Breen
G. D. Brown
J. D. Brown
R. A. Burr
S. H. Clark
R. M. Copeland
J. L. Dixson
C. E. Donnelly
Dom Echeverria
W. A. Farrow
C. M. Foster
M. L. Green
Harold Hammerquist
H. E. Hansen
A. B. Hayes
D. M. Hayes
R. E. Hughes
E. R. Jameson, Jr.
R. G. Jones
R. C. Keyser
H. G. Krog
S. E. Law
W. L. Lee
R. C. Leslie
W. J. Long
M. U. McKie
G. L. McMahan
J. R. McMurray
M. C. McSpadden
G. P. Malone
H. A. May
Wilmer Moore
C. L. Morrison
J. F. Munn
T. P. Murphy
F. P. Mursko
J. R. Neale
A. V. Ohlund
R. R. Porter
G. J. Puls
G. T. Purvance
Alton Reiten
E. H. Scheel
C. L. Seger
G. A. Severin
P. Wm. N. Smith
H. N. Snow
J. P. Sullivan
R. D. Surplus
R. M. Thackeray
R. W. Tillman
J. H. Tobiska
R. C. Tolley
R. W. Vernon

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Cornell University.—At the 1953 commencement exercises of Cornell University, 46 candidates were presented for the D.V.M. degree.

C. J. Boardman
E. B. Boroson
P. C. Buckley
W. C. Buell
R. L. Chandler
C. F. Clause
J. E. Croshaw, Jr.
L. R. Crowell
L. S. Dann
G. R. Davis
D. F. DiBitetto
R. P. Draudt
M. F. Ebersol
L. A. Goldfinger
L. W. Goodman
A. E. Greiner, Jr.
C. E. Hall
R. W. Harter
G. L. Hayes
P. E. Hoffman
S. H. Hull
J. H. Jacobson
F. T. Lewis
J. Lewis
A. O. Lindblom, Jr.
R. P. Lunna
W. W. Mead, Jr.
W. L. Mitchell
A. Motta
R. C. Nelson
L. O. Nezvesky
G. J. Plumer
F. W. Rapp
J. H. Robbins
Mary H. Rowan (Ford)
C. W. Sanderson
D. C. Schlomchug
A. L. Shor
F. A. Simpson
W. F. Stack
G. W. Sumner, Jr.
J. L. Thorne
W. J. Twining
W. J. Vineyard
R. D. Williamson
E. B. Winokur

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University of Georgia.—At the commencement exercises of the University of Georgia on June 8, 1953, 51 candidates were presented for the D.V.M. degree.

H. W. Armstrong
T. R. Barrett, Jr.
L. R. Bedwell
A. C. Benson, Jr.
J. J. Clark
Deron Collins
R. T. Copeland
E. A. Dobry
K. K. Dockery
N. S. Dorsey
F. W. Ellett
G. A. Elliott
W. H. Fletcher
T. E. Flowers
D. C. Forbes
O. M. Fulcher
Edward Garner
M. C. Gibbs
W. C. Hare
F. A. Hayes, Jr.
H. W. Helberg, Jr.
C. W. Henry
H. H. Holbrook
L. L. Howze
Fred Jones
W. H. Justice
J. A. Kimsey
A. L. Kleckner
V. A. Krabill
G. W. Loudenslager, Jr.
L. R. Luce
P. H. McMillan
N. W. Midyette
S. L. Moore
B. M. O'Kelley, Jr.
J. B. O'Quinn
C. B. Patton
W. G. Pearson
H. S. Powell
I. T. Rhodes
R. B. Rigler
M. S. Setser
F. H. Smith
G. L. Smith
H. E. Stinson
J. T. Tumlin
L. E. Van Petten
J. L. Veatch
B. Y. Ward
J. F. Wheeler
D. J. Williams, III

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University of Illinois.—At the 1953 commencement exercises of the University of Illinois, 22 candidates were presented for the D.V.M. degree.

F. F. Adams
Jerome Banicki
H. J. Brouwer
J. A. Buckler
P. E. Burkitt
B. D. Campbell
D. J. Carren
Gerald Dart
W. C. Dolowy
J. H. Dust
John Ehrhardt
R. K. Etheridge
R. W. Galvin
J. J. Grafly
J. J. Haxby, III
E. L. Kingry
K. H. Klingele
C. M. Linker
E. T. Owings
J. F. Palmer
L. F. Striegel
G. L. Waxler

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Iowa State College.—At the commencement exercises of the Iowa State College on June 12, 1953, 57 candidates were presented for the D.V.M. degree.

N. J. Anarumo
E. L. Anderson
R. H. Anthony
Peter Bendorf
John Bowne
R. D. Boyce
R. S. Boyer
E. M. Braun
R. J. Buckman
W. H. Bunting, Jr.
L. V. Carlyle
N. Y. Chung
E. L. Cook
C. E. Cotton
C. R. Denham
S. F. Dirks
R. F. Dougherty
G. R. Esbeck
D. O. Faulkner
W. D. Felker
R. E. Gamble
C. O. Gardner
D. D. Gillette
K. W. Gray
R. F. Hagedorn
J. M. Heacock
J. A. Hill
C. B. Inman
D. B. Jillson
R. D. Johnson
J. F. Krob
Robert Lapsley
L. L. Lemanski
R. M. McCully
W. P. McGrory
J. C. Marcum
R. G. Miesbauer
M. H. Moore
W. J. Neuliep
D. V. O'Connell
D. M. Pedigo
G. R. Petersen
H. J. Peterson
L. O. Rasmussen
D. W. Rubel
W. P. Sanders
F. E. Schneckloth
D. E. Smith
V. D. Spear
W. J. Spearing
C. J. Starch
C. O. Sudhoff
D. W. Thomas
R. J. Uhri
Warren Ver Ploeg
J. M. Wenzler
D. H. Wheeler

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Kansas State College.—At the commencement exercises of Kansas State College on May 24, 1953, 66 candidates were presented for the D.V.M. degree.

R. L. Adams
D. E. Awe
E. D. Baker
E. S. Beaver
W. F. Bradley
L. D. Breeden
J. J. Brennan
R. A. Claybaker
D. F. Cline
B. J. Edmundson
D. S. Edwards, Jr.
L. E. Fairbairn
B. R. Flack
J. D. Foote
L. A. Forsberg
T. M. Garrett
J. R. Good
A. P. Gray
Joanne Gross
M. A. Hammarlund
C. D. Heinze
A. B. Hillhouse
Ray D. Holder
Richard D. Holder
C. D. Hughes
J. H. Hurlburt
G. E. Hurley
W. G. Jones

C. A. Lindsay
R. S. Loomis
W. L. Lynn
J. F. Lyons
M. E. Martin
J. H. McCahon
C. F. McCannon
N. D. McCoss
E. S. McCreight
G. H. Merrill
G. J. Mitchell
D. L. Nelson
D. R. Nicholson
L. J. Russell, Jr.
L. V. Russell
J. D. Savage
J. F. Schindelar
W. R. Schlecty
J. R. Schnelle
D. F. Schwindaman
R. D. Simon
C. L. Smith
J. T. Smith
R. E. Smith
J. A. Stanberry
F. H. Stockton
R. J. Streeter
P. R. Strom
M. L. Swartz
S. S. Swift
R. S. Troutman
M. G. Watchorn
H. G. Warren
M. S. Watts
M. H. Werbin
L. A. Whitehair
G. A. Windisch
N. S. Wolf

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Michigan State College.—At the 1953 commencement exercises of Michigan State College, 50 candidates were presented for the D.V.M. degree.

R. S. Allen
R. D. Barnes
F. W. Binkley
T. A. Blackburn
I. L. Burness
J. S. Butts
P. S. Chaffee
R. E. Cook
M. L. Crandall
D. J. David
R. S. Dow
Harold Duchan
G. E. Gay
R. M. Gibbons
R. G. Haight
R. F. Hansen
P. L. Harrison
S. K. Harvey
Janet Howard
R. E. Kader
Simon Kalish
W. A. Kimball
E. J. Klein
Donald Konyha
E. J. Larson
F. O. May
J. R. Miller
L. E. Moffit
F. L. Newbury
R. W. Newlin
J. C. Nichols, Jr.
J. L. Norton
J. T. O'Connor
DeWitt Owen
M. J. Pettitt
Norman Piersma
T. E. Platt
Winthrop Ray
J. R. Reif
W. R. Rilett
J. C. Schlosser
H. D. Sheridan
K. W. Soncrainte
Donald Spotts
J. H. Stegeman
J. R. Tanzola
D. J. Thompson
H. Q. Tucker, Jr.
J. W. Watts
E. H. Wung

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University of Minnesota.—At the commencement exercises of the University of Minnesota on June 13, 1953, 48 candidates were presented for the D.V.M. degree.

D. K. Anderson
R. W. Bakke
A. C. Batchelder
M. A. Beck
E. N. Bergman
E. P. Bonde
G. D. Brakke
H. B. Cook
R. E. Dracy
N. S. Dzubay
D. B. French
E. E. Grass
H. H. Grote
D. D. Hagg
R. F. Hammer
J. O. Hanson
J. H. Hessian
B. F. Jahn
D. W. Johnson
V. P. Kelly
L. L. Larson
W. A. Leibbrand
Eugene Lindholm
K. I. Loken
D. F. Long
G. R. Lyon
P. J. McDermott
H. E. Meyer
W. C. Moberg
B. R. Nechay
L. S. Nelson
D. M. Oolman
R. F. Palmer
D. T. Pearson
W. M. Peden
G. W. Peterson
R. G. Peterson
D. E. Pietz
M. L. Pietz
F. E. Siegfried
R. H. Steinkraus, Jr.
E. M. Thom
L. W. Thom
W. M. Wass
R. F. Weidner
P. J. Whalen
S. J. Wilson
L. A. Zehrer

University of Missouri.—At the 1953 commencement exercises of the University of Missouri, 29 candidates were presented for the D.V.M. degree.

C. A. Berry
J. R. Bruner
W. F. Bryson
J. W. Counsilman
F. A. Elliott
H. C. Eschenroeder
J. R. Ganaway
R. G. Greeley
C. M. Hibbs
Theodore Higgins
D. N. Johnson
K. C. Kent
K. L. Knoernschild
W. W. Love
R. C. Manfull
A. M. Matteson
J. O. Minnick
R. A. Mutrux
P. L. Nelson
W. F. Noland
V. E. Owens
C. R. Plegge
H. H. Robertson
L. E. Rohlfing
A. P. Smith
J. Q. Thurmon, Jr.
R. D. Wallace
H. E. Wheatley
J. J. Woolsey

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Ohio State University.—At the commencement exercises of the Ohio State University on June 12, 1953, 64 candidates were presented for the D.V.M. degree.

N. D. Abbott
D. K. Adams
J. L. Anderson
H. V. Appleman
R. K. Arnold
R. W. Bachmeyer
F. E. Banks, Jr.
R. F. Behlow
J. H. Bennett
E. L. Bowlus
R. D. Burns
J. D. Coltrain
R. W. Copelan, Jr.
R. L. Craig
W. M. Culbertson
R. D. Daniels
R. A. Decker
H. G. Dresbach
A. J. Fleck
J. M. Gigliotti
N. C. Good
R. A. Griesemer
R. J. Haxby
M. E. Hays
J. J. Headlee
W. R. Hendricks
J. A. Hines
McClain Jackson
G. C. Janney
W. F. Keller
F. P. Killian
I. M. Lanning
R. W. Lybrook
M. E. Maxson
J. A. McClain
O. W. McClung
J. A. McVicker
F. D. Millar
Samuel Miller
J. R. Mitchell
H. R. Monfort
F. J. Mueller
E. G. Ongert
E. H. Page
S. W. Pawlowski
G. F. Pfeifer
K. I. Pittman
T. E. Powers
O. A. Ranstead
A. D. Rees
J. H. Russell
P. R. Schnurrenberger
L. E. Siddall
D. E. Smith
J. J. Spurgeon
R. F. Stryffeler
C. R. Taylor
E. R. Taylor
J. C. Taylor
J. R. Toth
W. J. Weber
R. D. Weidner
H. E. Wilkin
J. R. Wine

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Oklahoma A. & M. College.—At the 1953 commencement exercises of the Oklahoma A. & M. College, 30 candidates were presented for the D.V.M. degree.

W. L. Alberty
I. D. Alexander
C. W. Brown
T. A. Byrd
L. E. Carlin
C. D. Cason
D. E. Clymer
R. D. Conrad
J. P. Devine
J. M. Gambardella
F. W. Hester
M. L. Hill
K. W. Huffman
C. A. Kirkbride
L. J. Kimray
F. T. Lynd
S. L. Morrison
J. M. Oyler, Jr.
Alvin Pailet
R. J. Panciera
W. A. Potts
W. M. Pounds
L. D. Schwartz
R. H. Swayze
J. H. Venable
J. A. Walker
I. R. Wilcox
R. E. Williams
D. A. Woesner
A. V. Yanda, Jr.

University of Pennsylvania.—At the 1953 commencement exercises of the University of Pennsylvania, 40 candidates were presented for V.M.D. degree.

J. F. Basque
E. G. Blackburn
D. D. Bleicher
M. N. Brubaker
G. C. Bullock
D. B. Caldwell
H. J. Coleman
J. T. Cordell
R. N. Craft, Jr.
F. T. Custer, Jr.
W. L. Downey
H. C. Epperly
H. C. Fager
S. A. Fox
E. C. Garlick
E. J. Hathaway
A. P. Hollister
R. C. Horn
Klaus Hubben
P. W. Husted
J. P. Kreier
J. H. Landis
R. J. Lehman
R. S. Lescallette
A. M. Lowry
E. L. Loy
E. E. Marx, Jr.
J. O. Mason
J. P. McClure, Jr.
E. F. Neufer
T. J. O'Brien
A. G. Olivier
R. E. L. Probasco
S. C. Rice
Martin Sevoian
Albert Singer
J. D. Slick
J. F. Thompson, Jr.
W. A. Truban
S. L. Wolf

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Tuskegee Institute.—At the commencement exercises of Tuskegee Institute on May 18, 1953, 3 candidates were presented for the D.V.M. degree.

W. P. Higgins
T. A. Jeter
M. V. Weathersby

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State College of Washington.—At the 1953 commencement exercises of the State College of Washington, 41 candidates were presented for the D.V.M. degree.

D. B. Anderson
G. C. Baker
E. J. Brauner
C. W. Colenbaugh
E. L. Cooper
Robert Crossley
G. R. Ekeberg
J. W. Ferdinandsen
N. B. Gale, Jr.
A. H. Gardner
L. A. George, Jr.
Bill J. Gitzen
C. C. Guptill
R. J. A. Holte
E. W. Kettel
E. R. Klimke
R. E. Larson
R. G. Lewis
D. R. Marble
D. B. Martin
William Maxwell
R. C. McKnight, Jr.
J. W. Metcalf
M. A. Nilson
J. W. Norene
T. R. Pelley
J. W. Pickrell
Louis Polansky
C. M. Reed
R. D. Reid
F. N. Royal
H. W. Russell
J. A. Ryncarz
R. N. Smith, Jr.
M. D. Snook
R. J. Strong
C. G. Ulrich
H. W. Vaughn
P. J. Van Eeckhout
G. J. Venema
L. E. Wood

U. S. GOVERNMENT

Reorganization Plans for the Department of Agriculture Announced.—The new organization of the U.S.D.A., announced on Oct. 13, 1953, puts all of the service agencies of the Department under four main groups: (1) federal-state relations; (2) marketing and foreign agriculture; (3) agricultural stabilization; and (4) agricultural credit.

Under the proposal the research, inspection, disease, and pest control work now in the bureaus of the Agricultural Research Administration would be reorganized and regrouped—the research work in one group and the inspection and control work in another.

The administration of the Insecticide Act and the poultry meat inspection work would be moved from the Production and Marketing Administration to the new Agricultural Research Service.

The reorganization will not go into effect until there has been a period of public notice and until there has been further opportunity for comments from interested parties. The Intention Statement asks that such comments be made by Nov. 1, 1953.

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Veterinary Personnel Changes.—The following changes in the force of veterinarians in the U. S. Bureau of Animal Industry are reported as of Sept. 18, 1953.*

NEW APPOINTMENTS

Romualdas V. Buivid, Cincinnati, Ohio.
Carl E. Estes, National Stockyards, Ill.
Mark Field, Mexico City, Mex. (cancelled).
Forris B. Frick, Portland, Ore.
Calvin C. Gatz, San Francisco, Calif.
James Guess, Bismarck, N. Dak.
Robert J. Keller, Oklahoma City, Okla.
Robert P. McCoy, Jr., Denver, Colo.
John F. Manning, Kansas City, Kan.
Vytautis Maurutis, Cleveland, Ohio.
Wallace E. Mattson, Madison, Wis.
Alan G. Perkins, Sacramento, Calif.
Emerald G. Thomas, Jefferson City, Mo.
Mark H. Werbin, Wichita, Kan.

TRANSFERS

Hugh C. Beasley, from Raleigh, N. Car., to Mexico City, Mex.

Paul P. Boriskie, from Salt Lake City, Utah, to Lake Charles, La.

Robert G. Freel, from Boston, Mass., to Mexico City, Mex.

Leo F. Gordon, from Omaha, Neb., to Memphis, Tenn.

J. Howard Hathaway, from Portland, Ore., to Sioux City, Iowa.

Melvin J. Hatter, from Baltimore, Md., to Springfield, Mass.

William S. Houk, from Springfield, Mass., to Davenport, Iowa.

James L. Orr, from Mexico City, Mex., to Louisville, Ky.

Israel Saturen, from Indianapolis, Ind., to Lancaster, Pa.

Robert E. Scott, from Mexico City, Mex., to Louisville, Ky.

Ashley G. Stamp, from Detroit, Mich., to Cleveland, Ohio.

Martin L. Tuttle, from St. Louis, Mo., to Pittsburgh, Pa.

Howard L. Underwood, from Fort Worth to Houston, Texas.

Daniel F. Werring, from Mexico City, Mex., to St. Paul, Minn.

James P. Whitman, Jr., from Memphis, Tenn., to St. Louis, Mo.

H. Verne Wiser, from Salt Lake City, Utah, to Albuquerque, N. M.

*Space did not permit publication of "Resignations, Retirements, and Separations." They will be published in the December JOURNAL.

AMONG THE STATES AND PROVINCES

Alabama

Conference for Veterinarians.—The Alabama Polytechnic Institute School of Veterinary Medicine sponsored its twenty-ninth annual conference for veterinarians on Sept. 17-19, 1953.

The visiting lecturers included **Drs. J. C. Baker,** Birmingham; **G. R. Bartlett,** Atlanta, Ga.; **C. L. Blakely,** Angell Memorial Hospital, Boston, Mass.; **E. A. Davis,** Columbus, Ga.; **L. P. Doyle,** Purdue University, Lafayette, Ind.; **L. E. Johnson,** Ohio State University, Columbus, Ohio; **G. V. Kenmore,** Montgomery; **J. A. McCallam,** Washington, D. C., president of the AVMA; **G. J. Phelps, Jr.,** Montgomery; **S. J. Roberts,** N. Y. State Veterinary College, Cornell University, Ithaca; **B. T. Simms,** chief, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.; **D. D. Stamm,** Virus Laboratory, U.S.P.H.S., Montgomery; **J. W. Thome,** Atlanta, Ga.; **V. L. Wells,** Selma; and **H. T. Weir,** Birmingham.

Members of the School of Veterinary Medicine also participated in the demonstrations.

On Friday, members enjoyed a barbecue luncheon at the Saugahatchee Country Club.

s/R. S. SUGG, *Dean.*

California

Dr. K. F. Meyer Awarded Honorary Degree by University of Hannover.—Dr. Karl F. Meyer, director of the Hooper Foundation, University of California, was given the honorary degree of doctor of veterinary medicine by the veterinary faculty, University of Hannover, Germany, on the occasion of the 175th anniversary of the college, which was celebrated July 29-31, 1953. Dr. Meyer gave one of the two principal addresses on the program, the subject being "Preventive Veterinary Medicine." The citation which accompanied the degree read as follows:

> The Veterinary College of Hannover, under the presidency of Professor Dr. R. Völker, awards to Dr. K. F. Meyer, director of the George William Hooper Foundation for Medical Research, University of California, San Francisco, the degree of Doctor of Veterinary Medicine *honoris causa,* in appreciation of his outstanding merit in investigating and combating zoönoses and in appreciation of his world-wide and tireless advancement of scientific relations among nations.

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Central Association.—On Aug. 25, 1953, the Central California Veterinary Medical Association met at the Hotel Johnson in Visalia. The guest speaker was **Dr. William E. Maderious,** manager of the El Peco Ranch in Medera.

Seventeen members and seven guests attended the July meeting in Fresno. At this meeting, Dr. W. C. Truckell was voted an hororary membership and **Drs. Alex Robertson** and **John P. Hughes** reported on the highlights of the meeting of the state Association. **Drs. I. G. LaRue** and **W. W. Worcester** discussed the new state regulation on the handling of brucellosis reactors.

s/PAUL CARLSON, *Secretary.*

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Dr. Rosenberger Retires from Department of Agriculture.—Dr. Arthur C. Rosenberger (WSC '08), of Stockton, Calif., recently retired after forty years of service in California as senior field veterinarian of the state's department of agriculture. At a dinner at which he was the guest of honor, his many friends presented him with a check with which to buy a new automobile.

Canada

Report on Animal Diseases.—The August, 1953, report of the Health of Animals Division, Department of Agriculture, Canada, was quite favorable. Of the 16 reportable contagious diseases of domestic animals listed, outbreaks had occurred in only one; four premises were reported to be infected with typhoid.

Colorado

State Association.—The Colorado State Veterinary Medical Association held its annual fall meeting at Greeley on Sept. 10-11, 1953, with **Dr. K. V. Bolton** presiding and with about 100 veterinarians in attendance.

Out of state speakers included **Drs. A. H. Quin,** Kansas City; **Mark Morris,** Topeka, Kan.; **G. R. Moore,** East Lansing, Mich.; **K. W. Smith,** Sioux City, Iowa; and **W. A. Aitken** of Chicago. Home state speakers included **Dr. Rue Jensen,** Fort Collins and **Drs. John Mason** and **J. F. Winn,** U. S. Public Health Service, Greeley.

An excellent banquet with entertainment but without speeches was enjoyed Friday evening at the Greeley Country Club.

Officers elected to serve for the coming year are Dr. Arthur Herzberger of Colorado Springs, president, and Dr. W. R. Haas of Eaton, secretary.

District of Columbia

Death of Dr. H. E. Moskey.—Dr. Henry E. Moskey, 61, veterinary medical director of the Food and Drug Administration, died on Aug. 24, 1953, after a four-day illness.

Born at Hyattsville, Md., Aug. 14, 1892, Dr. Moskey received his early education in the District and his V.M.D. degree from the University of Pennsylvania in 1920. He entered government service as a messenger in the Bureau of Animal Industry in 1911 and later

served as clerk before entering the Army in World War I. After the war, he completed his professional training and returned to the Bureau where he did work in bacteriology and was later assigned to disease control work in Texas, California, and other areas.

He transferred to the Food and Drug Administration in 1927 and since 1928 had directed regulatory operations concerned with the safety, efficacy, and honest labeling of veterinary medical preparations.

Dr. H. E. Moskey

He was a member of the AVMA, having joined in 1920, and of the Association of Research Workers in Animal Diseases in North America. He lived at 924 South Twenty-Eighth Street, Arlington, Va.

Surviving Dr. Moskey are his widow, Louise Pollard, his son, a sister, and two brothers. Interment was at Arlington National Cemetery.

Dr. Moskey had served the AVMA for many years as representative to the U. S. Pharmacopoeial Convention and at the last convention in 1950 undertook, along with others, to obtain membership for a veterinarian on the important Committee on Revision.

Georgia

Personal.—First Lieutenant, V. C., Loyce W. Turner has been commanding officer of the 116th Medical Detachment in Bordeaux, France, since July 7, 1953.

Illinois

Northern Association.—On September 16, the Northern Illinois Veterinary Medical Association held its fall meeting at the Hotel Faust in Rockford.

The following speakers participated in the scientific program: **Drs. A. H. Brueckner,** Pitman-Moore Co.; **R. Leland West,** Waseca, Minn.; **T. H. Ferguson,** Lake Geneva, Wis.; **A. K. Merriman,** Sullivan; **V. W. Geselichen,** Omaha, Neb.; **W. O. Brinker,** Michigan State College, East Lansing; **R. C. Klussendorf,** Commercial Solvents Corp., Terre Haute, Ind.; **A. J. Legner,** Leland; and **F. E. Connor,** Morris.

s/J. W. BOLLER, *Secretary.*

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Chicago Association.—At the September 8 meeting of the Chicago Veterinary Medical Association, the following speakers comprised the program: **Drs. Harry W. Boothe,** who discussed diseases of the chinchilla; **A. Grant Misener,** who told of some of the activities of the annual meeting of the AVMA in Toronto; **Lester E. Fisher,** Berwyn, summarized the small animal television program at Toronto; and **O. Norling-Christensen,** Wilmette, reported on meetings of the Executive Board in Toronto.

On September 16, several interesting case reports were presented at the first fall meeting of the seminar group which met at the headquarters of the Anti-Cruelty Society.

s/WAYNE H. RISER, *Secretary.*

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Hog Cholera Virus Bill Defeated.—A bill to prohibit the use of virulent hog cholera virus in the State of Illinois after June 30, 1955, made some headway in the Illinois legislature but failed to pass on the third reading in the House on June 19, 1953. This leaves Alabama as the only state prohibiting the use of virulent hog cholera virus.

Indiana

Short Course for Veterinarians.—The forty-first annual short course for veterinarians, sponsored by Purdue University, was held Oct. 7-9, 1953.

In addition to the faculty members of the Department of Veterinary Science, the following speakers participated in the various sections: Large Animal Section—**Drs. L. E. Andres** of Remington, **C. R. Baumgartner** of Spencerville, **H. D. Carter** of Fairmount, **H. A. Lidikay** of Darlington, **C. L. Lindborg** of Monticello, **M. D. Neuhauser** of West Lafayette, **R. F. Portman** of Lafayette, and **P. F. Scott** of New Market; Swine and Sheep Section—**Drs. J. R. Davis** of Franklin, **K. N. Decker** of Brook, **L. W. Hinchman** of Glenwood, **A. L. Keim** of Kokomo, **E. W. Kline** of Thorntown, **G. R. Oldham** of Kokomo, and **T. L. Steenerson** of Wilkinson; Poultry Section—**Drs. L. A. Clark,** of Bedford, **M. M. Coble** of Columbia City, **R. M. Hafner** of Huntington, **D. S. Kline** of Boswell, and **F. C. Tucker** of Claypool.

Other guest speakers included **Drs. F. E. Connor,** Morris, Ill.; **J. W. Green,** state veterinarian, Indianapolis; **J. G. Hardenbergh,** Chicago, executive secretary of the AVMA; **T. K.**

Jones, U. S. Bureau of Animal Industry, Indianapolis; **T. J. King** (M.D.), health department, Chicago; **F. Kingma,** Ohio State University, Columbus; **A. A. Lenert,** A. & M. College of Texas, College Station; **P. P. Levine,** New York State Veterinary College, Ithaca; **George Scarseth,** Lafayette; **Richard E. Shope,** Rockefeller Institute, New York City; **A. C. Todd,** Department of Veterinary Science, University of Wisconsin, Madison; and **Jacob Traum,** U. S. Bureau of Animal Industry, Washington, D. C.

s/L. M. HUTCHINGS, *Head, Department of Veterinary Science.*

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Central Society.—The Central Indiana Veterinary Medical Society presented a round-table discussion on small animal problems at its October 28 meeting in Indianapolis. The meeting was opened by a detailed discussion on several subjects in the small animal field, followed by a smorgasbord at the Hotel Marrott, and then a general discussion of these problems by all practitioners in attendance. **Dr. George Burch,** Indianapolis, was moderator, and the panel members were **Drs. Grant Misener** and **Kenneth Bone** of Chicago, **Paul White** and **Bob Hoskins** of Indianapolis.

s/L. M. BORST.

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Michiana Association.—At the meeting of the Michiana Veterinary Medical Association in South Bend on September 10, **Dr. Alan Winter,** Benton Harbor, Mich., moderated a round-table discussion on nutrition in large and small animals.

s/J. L. KIXMILLER, *Resident Secretary.*

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Northwestern Association.—The Northwestern Indiana Veterinary Medical Association met at the Dr. R. F. Smith summer cottage on Lake Schaefer near Monticello on August 27. This was primarily a social meeting and members enjoyed boating, fishing, swimming, visiting, and barbequed chicken with all of the trimmings. The success of this meeting was due largely to the careful planning of Drs. Smith, Kline, and Krill and their wives.

s/J. L. KIXMILLER, *Resident Secretary.*

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Indiana-Illinois Association.—The Indiana-Illinois Veterinary Medical Association met at Gregg Park in Vincennes on September 18. **Dr. R. C. Klussendorf** of the Commercial Solvents Corporation in Terre Haute, Ind., discussed respiratory diseases of cattle. **Dr. M. R. Jollie,** Fort Branch, Ind., told of some problems of equine practice. A covered dish dinner was served at noon by the women.

s/J. L. KIXMILLER, *Resident Secretary.*

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Tenth District Association.—On August 20, members of the Tenth District Veterinary Medical Association met at the Greenfield Country Club in Greenfield to hear **Dr. L. C. Ferguson** of the Ohio State University discuss the importance of the laboratory to the practicing veterinarian.

Dr. G. T. Edds, Fort Dodge Laboratories, Fort Dodge, Iowa, spoke on better handling and treatment of mastitis in the dairy cow, at the meeting of the Association on September 17. Prior to the dinner and scientific meeting, members enjoyed trap shooting and playing cards.

s/J. L. KIXMILLER, *Resident Secretary.*

Iowa

Nutrition Conference.—The fourth nutrition conference for veterinarians was held September 10, 1953, at Curtiss Hall, Iowa State College, Ames. This conference was sponsored by the Feed Institute, Western Grain Feed Association, Iowa Veterinary Medical Association, and the Iowa State College.

The following speakers participated in the program: **Mr. Hugh Kelly,** Ames Reliable Products Co., Ames; **Mr. Laurence E. Carpenter,** executive director, Distillers Feed Research Council, Cincinnati, Ohio; **Dr. Norman Jacobson** (Ph.D.), **Dr. Wise Burroughs** (Ph.D.) and **Damon Catron** (Ph.D.), of Iowa State College; and **Dr. I. A. Merchant,** dean of the School of Veterinary Medicine and director of the Veterinary Research Institute, Iowa State College.

s/I. A. MERCHANT.

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Death of Dr. A. R. Menary.— Dr. A. R. Menary, 67, former city dairy inspector of Cedar Rapids for almost thirty years, died of cancer on Sept. 13, 1953. He had been ill for several months.

Born April 3, 1886, Dr. Menary received his degree in veterinary medicine from Iowa State College in 1910. He served for a brief period in World War I as a second lieutenant in the Veterinary Corps. Active in many organizations, he was a past president of the Iowa Veterinary Medical Association, the Eastern Iowa Association, of which he was treasurer for twelve years, and the East Central Iowa Association. He was also a past-president of the Linn County Tuberculosis Association and was president of the Scottish Society in 1953. He had been a member of the AVMA since 1932.

During his many years of service as dairy inspector, Dr. Menary effected many advancements in milk sanitation in Cedar Rapids. At the time of his retirement in 1951, he had brought about tuberculin testing, testing for brucellosis, compulsory pasteurization and, finally, a complete grade A status for the city's milk supply under U. S. Public Health Service Standards. For these accomplishments, he was honored at a special dinner tendered him

on retirement by Cedar Rapids dairymen and others interested in milk sanitation.

Surviving are his widow, the former Helen Orr Dunlop, a son, three brothers, and two sisters.

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Personals.—Dr. Walter L. Bierring (M.D.), of Des Moines, recently retired after sixty years of devoted medical service in Iowa and as health commissioner for the state for twenty years. His valued coöperation with Iowa veterinarians has long been appreciated.

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Mr. L. R. Fairall, who is public relations consultant for the AVMA, and his wife, left recently for a six-week visit to Europe. Mr. Fairall is head of the Fairall Advertising Co., Des Moines, Iowa.

Kansas

Central District.—On September 13, the Central District of the Kansas Veterinary Medical Association held a clinic in Abilene.

Dr. H. R. Hein, Clay Center, assisted by **Drs. F. M. Beam,** Washington, and **D. L. Nelson** of Wakefield, demonstrated the technique for flank spaying of heifers. **Dr. W. M. Theis,** Hillsboro, demonstrated the use of a metal detector in locating "hardware indigestion" in cattle, after which **Dr. M. L. Henrikson,** Emporia, performed a rumenotomy. **Dr. A. H. Gish,** Eldorado, talked on the questionable virtues of cosmetic surgery in show cattle, while demonstrating some of his techniques, assisted by his son, **Dr. John Gish,** and by **Dr. J. F. Neal** of Winfield.

Dr. M. L. Twiehaus, newly appointed head of the Department of Pathology, Kansas State College, discussed some recent cases of atrophic rhinitis and demonstrated lesions on fresh specimens. **Dr. J. S. Haley,** Topeka, president of the state Association, demonstrated his technique of ear trimming. **Dr. F. W. Jordan,** Abiline, vice-president of the state Association, performed a tonsillectomy on a Greyhound.

While the men were enjoying the clinic, the women occupied their time at cards and gossip at Frank's club, where the men joined them for a fried chicken dinner.

The meeting was a record-breaker for district meetings in Kansas, with about 130 in attendance. At one time during the afternoon, there were about 80 veterinarians attempting to witness the proceedings. At the dinner, 96 were served in two shifts.

Dr. and Mrs. F. W. Jordan and Dr. and Mrs. G. W. Carl, of Abiline, were hosts to the group and are to be warmly congratulated for a most enjoyable time.

s/K. M. CURTS, *Resident Secretary.*

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Sedgwick County Association.—On September 10, the Sedgwick County Veterinary Medical Association met in the Stockyards Hotel in Wichita.

The program included a talk by **Dr. M. W. Osburn,** extension veterinarian at Kansas State College, on the nature of his duties; pictures and a commentary by **Dr. E. J. Frick** on his recent European tour; and a discussion led by **Dr. J. E. Feiser,** president of the association, on the various aspects of dog shows.

The following officers were elected to serve the association during 1954: Drs. James S. Hervey, Wichita, president; Harvey J. Hensley, Wichita, vice-president; and Ernest L. Boley, Wichita, secretary-treasurer. Drs. Joe A. Bogue and Leroy E. Dietrich, Jr., both of Wichita, were elected directors.

s/K. M. CURTS, *Resident Secretary.*

Massachusetts

State Association.—The regular monthly meeting of the Massachusetts Veterinary Association was held at the Highland Hotel in Springfield on September 23. The guest speaker was **Dr. Gerry B. Schnelle** of the Angell Memorial Animal Hospital, Boston, who discussed radiographic diagnosis of the abdominal mass.

s/C. LAWRENCE BLAKELY, *Secretary.*

Michigan

Southeastern Association.—On September 24, the Southeastern Michigan Veterinary Medical Association met at the Herman Kiefer Hospital in Detroit. The program included a film on fractures of the elbow which was narrated by **Drs. E. Oja** of Ferndale and **J. P. McEvoy** of Royal Oak. Films on blood banks and antibiotics were also shown.

s/GILBERT MEYER, *Secretary.*

Minnesota

Short Course.—The annual short course for veterinarians, sponsored by the University of Minnesota School of Veterinary Medicine was held Aug. 27-28, 1953, at University Farm, St. Paul, with 107 veterinarians in attendance.

In addition to University of Minnesota staff members, the following speakers participated in the program: **Drs. L. T. Christensen,** Hancock; **J. D. Ray,** director, Affiliated Laboratories, Corp., White Hall, Ill.; **L. J. Redder,** Canby; **H. Stoenner,** U. S. Public Health Service, Rocky Mountain Laboratory, Hamilton, Mont.; and **D. E. Trump,** Owatonna.

s/H. J. GRIFFITHS, *Resident Secretary.*

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Twin City Society.—The Twin City Veterinary Medical Society met in St. Paul on September 10. The guest speaker, **Mr. Robert Speer,** North Oak Kennels, presented an interesting and informative talk on training retrievers.

At the business session, twelve new members were admitted to the Society and the following officers were elected: Drs. W. N. Peterson, president; E. Fitch, first vice-president; H. H. Hoyt, second vice-president; and G. W. Mather, secretary-treasurer.

s/HENRY J. GRIFFITHS, *Resident Secretary.*

Missouri

Northeast Association.—The Northeast Missouri Veterinary Medical Association held a clinic meeting at the hospital of Hinds and son, Palmyra, on August 13. Participating in the clinic were: **Drs. E. F. Ebert,** director of clinics, University of Missouri School of Veterinary Medicine, Columbia; **M. S. Campbell** and **R. L. Campbell,** Brookfield; **H. D. Rodabaugh,** Kirksville; and **G. N. Haigler,** St. Louis. About 25 veterinarians and their families were present.

s/A. H. GROTH, *Resident Secretary.*

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Central Association.—On August 7, the Central Missouri Veterinary Medical Association had a picnic at the Memorial Park in Marshall. About 30 veterinarians and their families attended. A round-table discussion was held on current practice problems.

s/A. H. GROTH, *Resident Secretary.*

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Southeast Association.—The Southeast Missouri Veterinary Medical Association met at the Pemiscot County Veterinary Hosiptal of Dr. J. V. Moore at Hayti on September 16.

Those who participated in the program were **President Howard E. Dickman,** Perryville; **Drs. A. R. Bott,** East St. Louis, Ill.; **W. R. Lawrence,** Dyersburg, Tenn.; **W. L. Stroup,** Corinth, Miss.; **H. W. Howell,** University of Missouri, Columbia; and **L. A. Rosner,** state veterinarian, Jefferson City.

Members enjoyed a luncheon of old-fashioned southern pit barbecue and Mississippi River cat fish.

s/A. H. GROTH, *Resident Secretary.*

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Kansas City Association.—The Kansas City Veterinary Medical Association met in the Continental Hotel on September 15.

Dr. Warren Kilpatrick of Mediapolis, Iowa, discussed his approach to the many problems of a busy general practice.

s/J. C. DAVIS, *Secretary.*

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Dr. Stanley Smith Honored.—Dr. Stanley Smith (NYC '92), lecturer and clinician at the School of Veterinary Medicine, University of Missouri, was presented with a scroll at the recent Boone County Fair Horse Show. The scroll carried a citation commending Dr. Smith for his long and active interest in and support of the show. Dr. Smith is still active although he will be 83 on Nov. 30, 1953, and still serves as one of the ambulatory clinicians. He keeps informed on developments in his profession by reading current literature and regular attendance at state and national meetings. Truly, he is a remarkable citizen and veterinarian.

s/A. H. GROTH, *Dean, School of Veterinary Medicine.*

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Dr. Blake Joins Veterinary Faculty.—Dr. W. P. Blake (COL '41) has been appointed assistant professor of veterinary medicine and surgery at the School of Veterinary Medicine, University of Missouri, Columbia.

After receiving his D.V.M. degree, Dr. Blake spent four years on disease control work in Washington. Since 1945, he has been in general practice at Greeley, Colo., with his father, Dr. W. L. Blake.

s/A. H. GROTH, *Resident Secretary.*

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Animal Disease Incidence.—A report on animal diseases in Missouri for the six months ending June 30, 1953, revealed, among others, the following incidence: cattle diseases—anaplasmosis 119, anthrax 2, leptospirosis 54, listeriosis 85, mycotic stomatitis 70, x disease 22, and calf diphtheria 28; swine diseases—hog cholera 4,474 herds, swine erysipelas 2,444, atrophic rhinitis 1,196, transmissible gastroenteritis 1,076, anthrax 33, and eperythrozoonosis 228; sheep diseases—389 cases of sheep scab and 38 of listeriosis. A total of 190 cases of rabies was reported, including 55 in large animals: cattle 49, horses 3, swine 2, and sheep 1. There were 102 cases in dogs, 18 in cats, and 13 in furbearing animals.

New York

New York City Association.—The regular meeting of the Veterinary Medical Association of New York City, Inc., was held October 7 at the New York Academy of Sciences. The guest speaker was **Dr. John T. McGrath,** assistant professor of pathology at the School of Veterinary Medicine, University of Pennsylvania, Philadelphia. He discussed spinal paralysis of the dog, with illustrations.

Drs. R. L. Burkhart, R. J. Garbutt, and R. Meisels comprised the program committee.

s/C. E. DECAMP, *Secretary.*

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Dr. C. R. Schroeder Accepts Managing Directorship of San Diego Zoo—Dr. C. R. Schroeder, who has been manager of the animal industry section of Lederle Laboratories Division, American Cyanamid Company, with headquarters at Pearl River, N. Y., for a number of years, has accepted the position of managing director of the San Diego (Calif.) Zoo and took over his new duties in October.

Dr. Schroeder is well known in zoölogical park circles having been veterinary pathologist for the San Diego Zoological Society from

1933 to 1937, veterinarian of the New York Zoological Park from 1937 to 1939; he then returned to work with the San Diego Zoo in 1939-1941 before going with Lederle Laboratories in the latter year as assistant director of their animal industry section. For several years, he has served as secretary of the Veterinary Medical Association of New York City.

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Mr. Wentworth Honored.—Mr. W. A. Wentworth, who for years has presented the Borden Award annually to a member of the veterinary profession at the annual meeting of the AVMA, was the recipient of an award at Iowa State College. On June 13, 1953, he received the Chicago Merit Award for outstanding service in the advancement of human welfare. Mr. Wentworth graduated from the Iowa State College in 1910. He has been in the public relations service of the Borden Company of New York since 1929.

North Carolina

Officers of Central Carolina Association.—At the September 9 meeting of the Central Carolina Veterinary Medical Association, **Dr. H. J. Rollins,** state veterinarian, spoke on calfhood vaccination. The following officers were elected: Drs. J. T. Dixon, Winston Salem, president; J. A. Jordan, Lexington, vice-president; J. P. Ellis, Greensboro, secretary; and F. P. Wise, Greensboro, treasurer.

S/CLYDE W. YOUNG, *Resident Secretary.*

Ontario

Death of Dr. A. F. Bain.—Dr. Alexander Francis Bain, head of the Department of Bacteriology at the Ontario Veterinary College, suffered a heart attack and died suddenly on Aug. 27, 1953, at his home, 28 Liverpool St., Guelph, Ont. He is survived by his widow, the former Norma Luxton, and three children, David, Susan, and Andy. He was 41 years of age, and the son of Mr. and Mrs D. W. Bain of 127 Northumberland St., Guelph. A brother, David Bain, resides in Toronto.

Dr. Bain graduated from the Ontario Veterinary College in 1933 and joined the faculty of Macdonald College, Quebec. In 1938, he became a member of the faculty of the Ontario Veterinary College, and in 1941 was granted leave of absence to join the Canadian Army. On completion of his army training, he received the rank of lieutenant and was appointed instructor in the artillery wing at the Officers' Training Center at Brockville, Ont. Later, Dr. Bain became an instructor at A2 Artillery Training Center, Petawawa, Ont., from where he proceeded overseas as an artillery officer.

In recognition of Dr. Bain's ability as a bacteriologist, he was transferred in England to the Canadian Army Medical Corps. As an officer of No. 14 General Hospital Unit of this corps, he served in England and in the Mediterranean theater where he survived an enemy action in which the ship transporting the hospital unit was torpedoed and sunk. Dr. Bain returned to Canada and received discharge from the Army in 1945.

Dr. A. F. Bain

Shortly after rejoining the staff of the Ontario Veterinary College, Dr. Bain was appointed professor and head of the Department of Bacteriology, and held that position until his death.

Dr. Bain was a member of the U. S. Livestock Sanitary Association, the New York Academy of Sciences, the Canadian Public Health Association, the Society of American Bacteriologists, the Canadian Society of Microbiologists, the American Veterinary Medical Association, the Canadian Veterinary Medical Association, and the Ontario Veterinary Association.

He was a member of St. George's Anglican Church, Guelph, and an officer in the local committee of the Boy Scouts Association, and had formerly been secretary. Dr. Bain was also active in the little theater movement in Guelph.

Dr. Bain's understanding of the field of veterinary bacteriology was broad. He pioneered many aspects of veterinary research in this field. His influence was great. It will continue to exert its effect through his associates and many students who knew him as a teacher, counsellor, and friend.

Pennsylvania

State Association.—The seventy-first annual convention of the Pennsylvania State Veterinary Medical Association was held in the Hotel Roosevelt in Pittsburgh, Oct. 14-16, 1953.

The following speakers comprised the literary program: **Drs. John T. McGrath,** University of Pennsylvania, Philadelphia; **James Archibald,** Ontario Veterinary College, Guelph; **L. E. Fisher,** Berwyn, Ill.; **V. L. Tharp,** Ohio State

University, Columbus; **T. Y. Tanabe,** Pennsylvania State College, State College; **C. J. Hollister,** Montrose; **Brig. Gen. J. A. McCallam,** Washington, D. C., president of the AVMA; **Drs. S. F. Scheidy,** Drexel Hill; **Carl W. Groppe,** Elm Grove, W. Va.; **P. P. Levine,** New York State Veterinary College, Ithaca; **Mr. J. Lewis Williams,** Uniontown; **Drs. Gerald Fouse,** Zelienople; **H. S. Forney,** Palmyra; **W. R. Korns,** Somerset; **J. G. Eagelman,** Womelsdorf; **J. J. Connolly,** Indiana, Pa.; **A. V. Bartenslager,** Stewartstown; **W. P. Bond,** Bloomsburg; **H. H. Keeler,** Athens; **H. M. Mershon,** Linesville; **W. O. Brinker,** Michigan State College; **Mark W. Allam,** dean, School of Veterinary Medicine, University of Pennsylvania; **C. P. Markle** (M.D.), West-Penn Hospital, Pittsburgh; **F. J. Mulhern,** in charge, vesicular exanthema eradication program, U. S. BAI, Washington, D. C.; **H. A. Milo,** director, BAI, Harrisburg; **George Bennett,** Pittsburgh; **Blakey T. Deal,** Lansdowne; **Harry M. Hershman,** Uniontown; **Henry R. Becker,** York; **James T. Herron,** Canonsburg; and **Alan Bachrach,** Philadelphia.

s/RAYMOND C. SNYDER, *Secretary.*

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Bucks-Montgomery Association.—The Bucks-Montgomery Veterinary Medical Association met at the Moose Home in Doylestown on September 9. Members enjoyed a round-table discussion of the revision of the constitution and by-laws of the state and local associations and of the state mastitis program.

s/VINCENT W. RUTH, *Secretary.*

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Keystone Association.—Members, and their families, of the Keystone Veterinary Medical Association enjoyed their annual picnic at the New Bolton Center, London Grove, on Sunday, September 27.

s/ERNEST K. STEARLY, *President.*

Virginia

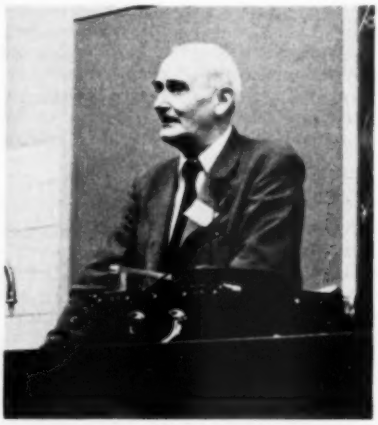
State Association.—The Virginia State Veterinary Medical Association held its semiannual meeting at the Cavalier Hotel, Virginia Beach, on September 10-12, with 93 members and guests in attendance.

The following speakers presented papers at the scientific session: **Drs. F. E. Mullen,** poultry consultant, Shen-Lab., Inc., Harrisonburg; **S. L. Kalison,** director, Virginia Artificial Breeding Association, Inc., Rocky Mount; **A. E. Hancock,** in charge, State Diagnostic Laboratory, Ivor; **W. F. Witter,** Richmond; **Brigadier General J. A. McCallam,** Washington, D. C., president of the AVMA; **Eric F. Pallister,** School of Veterinary Medicine, University of Pennsylvania, Philadelphia; **Wilson B. Bell,** Virginia Polytechnic Institute, Blacksburg; **A. H. Craige, Jr.,** chief veterinarian, Veterinary Medical Department, Pitman-Moore Company, Indianapolis, Ind.; **I. I. Franklin,** Emporia; **J. E. Lippincott,** Lynnhaven; and **W. L. Bendix,** state veterinarian, Richmond.

During the business session, fourteen new members were admitted. The next meeting will be held in conjunction with the West Virginia Veterinary Medical Association; the date has not been set.

s/A. J. SIPOS, *Secretary.*

Wisconsin



Dr. F. W. Schofield, Guelph, Ont., who discussed cattle diseases at the fifth annual Wisconsin postgraduate conference for veterinarians at the University of Wisconsin, June 8-10, 1953.

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Dr. and Mrs. Burr A. Beach at the conference dinner held during the postgraduate veterinary conference at the University of Wisconsin, June 8-10, 1953.

West Virginia

Personal.—Dr. Isaac H. Maxwell, Lost Creek, was elected secretary of the state Veterinary Board at a meeting of the board at Clarksburg on Aug. 17, 1953. He replaces Dr. W. E. Trussell.

FOREIGN NEWS

India

Free Medical Aid for Animals.—The Society for the Prevention of Cruelty to Animals in Delhi plans to register pack animals at their hospital and charge the owners a small monthly sum as a sort of hospital insurance on their animals. This is to encourage owners in the timely care of their animals. While an animal is in the hospital, it would receive free medical treatment and food.—*The Hindustan Times, July 8, 1953.*

S/MAJOR BENJAMIN FREMMING, V. C., *U.S.A.F.*

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Veterinarians to Have Use of Sulfonamides.—Veterinary practitioners have been debarred from prescribing sulfonamides and barbiturates, even when the treatment of an animal required the use of these drugs. Under the provisions of the drug act of 1940, only "registered practitioners" are permitted to prescribe these drugs, and veterinarians have never been registered as such. However, steps are now being taken to prepare such a register of veterinarians and it is hoped that soon they will have the same privileges in using these drugs as do the medical practitioners.—*The Hindustan Times, July 6, 1953.*

S/MAJOR BENJAMIN FREMMING, V. C., *U.S.A.F.*

Germany

Professor Kurt Wagener Awarded Honorary Degree in Medicine by University of Gottingen.—Dr. Kurt Wagener, professor and director of the Institute of Hygiene of the Veterinary College of Hannover University, was awarded the degree of doctor of medicine, *honoris causa*, by the faculty of medicine of the University of Göttingen, Germany, during the 175th anniversary of the veterinary college at Hannover which was celebrated July 29-31, 1953. The presentation was made by Professor Schütz, director of the institute of hygiene at Göttingen University.

Foreign Veterinarians and Animal Husbandrymen Visit in Wyoming

Veterinarians and animal husbandrymen from Turkey, Iran, Afghanistan, and Bolivia spent some time in Wyoming under the auspices of the Mutual Security Agency. A part of the group visited the Wyoming State Veterinary Laboratory. They are (front row, left to right)—Huseyn Elbasi, Kemal Demal, Naci Opren.

Second row—Hasip Kurtpinar, Sait Abusoglu, Urga, Huseyn Civelikoglu, Hasan Turker, Ihsan Atamyilmaz, and Fikri Cizmen.

Third row—Enver Erlat, Sodettin Unal, Salih Zek Ileri, Salahattin Yazgan, Omer Gebelek, Barrett, Glenn, and J. Weldon of the University of Wyoming. Two men from Afghanistan are unidentified.

In awarding the degree, Professor Schütz emphasized Dr. Wagener's contributions to studies on tuberculosis, milk hygiene, and public health.

Professor Wagener is well known in many

Dr. Kurt Wagener

countries, including the United States where he visited in 1951. For several years, he has been active in the German Association of Hygiene and Microbiology, having served as president, and as chairman of the veterinary section.

VETERINARY MILITARY SERVICE

Change in Office of Army Surgeon General.—On Aug. 25, 1953, Lieutenant Colonel William S. Gochenour, V.C., reported to the Office of the Army Surgeon General as chief of the Liaison, Standards and Animal Branch of the Veterinary Division. He succeeds Lieutenant Colonel Karl H. Willers, who entered the Army Medical Service Graduate School at Walter Reed Army Medical Center to attend the course in military preventive medicine.

Colonel Gochenour was a prisoner of war of the Japanese during World War II. In 1945, he was returned to the United States and served at the Cavalry Replacement Training Center at Fort Riley, Kan., and completed a course at the General Command and Staff School at Fort Leavenworth, Kan.

He was ordered to China in 1946 to serve with the Military Advisory Group. The Republic of China awarded him the Order of the Cloud and Banner for his work there. Upon his return, Colonel Gochenour was sent to Walter Reed Army Medical Center in December, 1950. At the time of his present transfer he was chief of the Department of Bacteriology of the Army Medical Service Graduate School and deputy chief of the school's veterinary division. Colonel Gochenour is a diplomate of the Board of Veterinary Public Health. He is married, has one daughter and makes his home in Silver Spring, Md.

Colonel Willers, who is from Nebraska, has been on duty in the Office of the Army Surgeon General since 1951. He began his career in the Army Veterinary Corps in March, 1934.

He served in the Far East for three years as assistant public health officer in the Civil Affairs Section, Supreme Command Allied Powers. Returning to the United States, Colonel Willers was a student at the Medical Field Service School at Fort Sam Houston, Texas, in the Medical Service Officers Advanced Course. In November, 1950, he was sent to the Office of the Army Surgeon General. He is married, has three children and makes his home in Arlington, Va.

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Colonel Miller Gets New Assignment.—Colonel Robert R. Miller, V.C., U.S.A.F., recently graduated from the Air War College, Air University, Maxwell Air Force Base. This college, offering the highest level military education of all schools operated by the Air Force, prepares carefully selected officers for command and key staff assignments. Colonel Miller, first veterinary officer to attend the Air War College, was the veterinary inspector general for the Air Force prior to attendance. His new assignment is at headquarters Air Defense Command, Colorado Springs, Colo., as the command veterinarian.

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Captain LeJeune Awarded Bronze Star.—On July 31, 1953, Col. J. W. Donnell, commanding officer, Kansas City Records Center, presented the Bronze Star Medal to Captain John E. LeJeune, Jr., for distinguished service as meat and dairy products food inspector in Japan from May 24 to Dec. 21, 1951. The citation read, in part ". . . His notable achievements reflect credit upon himself and the military service."

s/CAPTAIN RODNEY S. BILLETT, V.C.

Colonel J. W. Donnell (back to camera) presents the Bronze Star Medal to Captain John E. LeJeune, Jr. Others at the presentation were (left to right)—Second Lieutenant Joe M. Word, First Lieutenant Richard M. Lasday, First Lieutenant Lewis J. Runnells, Major James E. Peck, Captain Rodney S. Billett, Captain William R. Streeter, and Captain Keith P. Keller.

The Eighty-Second Class of Veterinary Officers Who Have Completed the Course at the Army Medical Service Meat and Dairy Hygiene School in Chicago

During the past two years, each class has visited AVMA headquarters. To date, 409 of these officers have inspected the central office facilities and heard talks on the work of the Association. Those in the picture are:

Front row (left to right)—First Lieutenant Elmer L. Robinson, instructor; Captain Harlan M. Stanton, instructor; Major Thomas A. S. Hays, instructor; Lieutenant Colonel Harry R. Lancaster, commandant; Major Edward P. Hornickel, instructor; Lieutenant Colonel George D. Batcheldor, director; First Lieutenant John A. Postle, adjutant; Captain John S. Zwiers, instructor.

Second row—First Lieutenant William D. Donovan, Captain Leonard D. Smith, First Lieutenant Harold M. Braeutigam, Second Lieutenant Howard F. Lancaster, Captain Allen N. Lang, Second Lieutenant Anthony W. Riepma, Captain Ray M. Hull, First Lieutenant James P. Amy, Jr.

Third row—Second Lieutenant Albert C. Batchelder, First Lieutenant George Drinas, First Lieutenant William R. Strieber, Captain Julius W. Amsiejus, Second Lieutenant Angus W. Gaskin, Second Lieutenant James F. Jordan; Captain Robert F. Burch, Captain Robert D. Thornberry.

Back row—Second Lieutenant George E. Gay, First Lieutenant Homer J. Moore, Captain Wesley H. Wertz, First Lieutenant John W. Siemens, Second Lieutenant Edward W. Speer, First Lieutenant Robert W. Barnett, First Lieutenant Joseph H. Gainer, Second Lieutenant Thaddeus W. Howard, First Lieutenant Jean N. Archer.

BIRTHS

Dr. (COL '53) and Mrs. R. C. Keyser, Ronan, Mont., announce the birth of a daughter, Penny Rae, on July 30, 1953.

Dr. (KSC '46) and Mrs. George T. Woods, Urbana, Ill., announce the birth of a son, David Jordan, on Aug. 14, 1953.

Dr. (SYD '52) and Mrs. Peter T. Diplock, Eltham, New Zealand, announce the birth of twin daughters, Lynda Margaret and Debra Jane, on June 10, 1953.

Dr. (TEX '43) and Mrs. Harry J. Magrane, Mishawaka, Ind., announce the birth of a

daughter, Jean, on Sept. 7, 1953. The Magranes also have two sons.

Dr. (COL '48) and Mrs. James G. Hardenbergh, Rockton, Ill., announce the birth of a son, John Gerard, III, on Oct. 2, 1953.

DEATHS

★**Alexander F. Bain** (OVC '33), 42, Guelph, Ont., died Aug. 27, 1953. Dr. Bain was admitted to the AVMA in 1939. An obituary appears on page 481 of this JOURNAL.

★**Allan J. Boddy** (WSC '50), 30, Salem, Ore., died July 9, 1953. Dr. Boddy was in general practice in Salem until March, 1953, when he entered the U. S. Bureau of Animal Industry as meat inspector in Salem. On July 1, 1953, he left the BAI to begin training for missionary service at the New Tribes Mission, a nondenominational school at Fouts Springs, Calif. When a fire broke out on July 9 in a nearby national forest, he volunteered to fight it and was one of the fifteen men trapped in a canyon and burned to death when the blaze got out of control and overtook them.

Dr. Boddy is survived by his widow and three daughters. He was a member of the AVMA.

Algie E. Campbell (CVC '15), 64, Rantoul, Ill., died Aug. 30, 1953. Dr. Campbell was a member of the Illinois State Veterinary Medical Association and had been a member of the AVMA.

★**Lieutenant Colonel Robert W. Cook** (KSC '36), 40, V.C., U. S. Army, died Sept. 21, 1953, from poliomyelitis following a brief illness. At the time of his death, he was stationed in New York City as quartermaster inspection service representative with the New York Quartermaster Market Center. Colonel Cook entered the military service in 1937 and except for a brief period had served continuously on active duty. He was commissioned in the Regular Army Veterinary Corps in 1946. He had only recently returned from a three-year tour of duty in the Far East, part of which was served in Korea.

He was twice awarded the Commendation Ribbon for outstanding service. Colonel Cook is survived by his widow and two children. Funeral services were held in Kansas City, Mo.

Colonel Cook was admitted to the AVMA in 1939.

William H. Eberle (ONT '14), Silex, Mo., died July 15, 1953. Dr. Eberle was for several years on the field force of the Bureau of Animal Industry in Missouri. He had practiced at Silex since 1950.

★ Indicates members of the AVMA.

★**Harry Hinds** (KSC '09), 69, Palmyra, Mo., died Sept. 10, 1953. Dr. Hinds had practiced in Palmyra for thirty-four years. He is survived by his widow and four sons, one of whom, Dr. Eugene Hinds (MO '52), was associated with him in practice.

Dr. Hinds was a member of the AVMA.

Earl R. Hunter (GR '12), 64, Detroit, Mich., died Jan. 31, 1953. Dr. Hunter had served as a lieutenant in the Veterinary Corps, U. S. Army, during World War I. He was a meat inspector for the Detroit Board of Health for eight years. In 1939, he established a small animal practice in Detroit, in which he was engaged until the time of his death.

★**Leslie E. Johnson** (OSU '30), 47, Rushville, Ill., died Aug. 2, 1953, of a heart attack while vacationing near Brainerd, Minn. Dr. Johnson was a member of the Mississippi Valley and Illinois State Veterinary Medical Associations and of the AVMA.

Martin F. Kent (MCK '18), 57, Chicago, Ill., died in September, 1953. Dr. Kent had recently retired as track veterinarian, but continued his practice. He is survived by his widow, four brothers, and two sisters.

★**Albert R. Menary** (ISC '10), 67, Cedar Rapids, Iowa, died Sept. 13, 1953. An obituary appears on page 478 of this JOURNAL. Dr. Menary was admitted to the AVMA in 1932.

★**Henry E. Moskey** (UP '20), 61, Washington, D. C., died Aug. 24, 1953. An obituary appears on page 477 of this JOURNAL. Dr. Moskey was a member of the AVMA.

Charles W. Sauer (CVC '08), 69, Vinton, Iowa, died July 5, 1953. Dr. Sauer had served as assistant state veterinarian for four years and as councilman for Vinton from 1933 to 1938. He is survived by a son.

★**Mellis G. Talbert** (IND '15), 69, Franklin, Ind., died during the early part of August, 1953. Dr. Talbert was a general practitioner. He was admitted to the AVMA in 1949.

★**John W. Watt** (OVC '23), 60, West Haven, Conn., died Sept. 4, 1953. Dr. Watt, a general practitioner, was a member and past president of the Connecticut Veterinary Medical Association. He was admitted to the AVMA in 1929.

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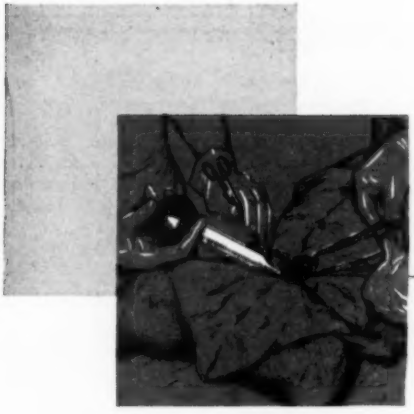
Death Reported in Error.—The report of the death of Dr. Randall W. Devereux in the October issue of the JOURNAL was in error. This information, given erroneously to the Ontario resident secretary, was reported to the central office. While we regret the error, we are happy that the information was not correct.—ED.



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COMING MEETINGS

Notices of Coming Meetings must be received by 4th of month preceding date of issue

Mississippi Valley Veterinary Medical Association. Annual meeting. Hotel Pere Marquette, Peoria, Ill., Nov. 4-5, 1953. A. C. Gathmann, 202 Front Street, Galva, Ill., secretary.

Omega Tau Sigma Fraternity. Annual convention. University of Georgia, Athens, Ga., Nov. 6, 1953. E. D. Risher, Jr., Box 235, Ag Hill, Athens, Ga.

Conference of Public Health Veterinarians. Hotel New Yorker, New York, N.Y., Nov. 9, 1953. Dr. James Lieberman, U. S. Public Health Service, Washington 25, D. C., secretary.

Association of Military Surgeons. Annual meeting. Statler Hotel, Washington, D. C., Nov. 9-11, 1953. Winchell McK. Craig, Rochester, Minn., president.

Southern Veterinary Medical Association. Annual meeting. Atlanta Biltmore Hotel, Atlanta, Ga., Nov. 9-11, 1953. A. A. Husman, Raleigh, N. Car., secretary.

Midwest Small Animal Association, annual meeting, and American Animal Hospital Association, regional meeting. Hotel Burlington, Burlington, Iowa, Nov. 18-19, 1953. J. Porter Coble, 2828 S. MacArthur Blvd., Springfield, Ill., secretary.

Nebraska Veterinary Medical Association. Annual meeting. Lincoln, Neb., Dec. 1-3, 1953. G. A. Ackerman, 319 L St., Lincoln, Neb., chairman, program committee.

Animal Care Panel. Annual meeting. Billings Hospital, University of Chicago, Chicago, Ill., Dec. 2-3, 1953. The program will be ready for distribution in September and may be obtained by writing to Robert J. Flynn, Secretary, Animal Care Panel, Argonne National Laboratory, P. O. Box 299, Lemont, Ill.

Arizona Veterinary Medical Association. Annual meeting. Phoenix, Ariz., Dec. 3-4, 1953. G. S. Calderwood, Chandler, Ariz., secretary.

Kentucky Veterinary Medical Association. Short course for veterinarians. Department of Animal Pathology Building, University of Kentucky, Lexington, Ky., Dec. 2-3, 1953. T. J. Stearns, Room 216, Live Stock Exchange Bldg., Louisville, Ky., secretary.

Maryland State Veterinary Medical Association. Annual winter meeting. Lord Baltimore Hotel, Baltimore, Md., Dec. 4-5, 1953. John D. Gadd, Cockeysville, Md., secretary.

University of Pennsylvania. Annual conference for veterinarians. University of Pennsylvania, School of Veterinary Medicine, Philadelphia, Pa., Jan. 5-6, 1954. Walter E. LaGrange, chairman, conference committee.

New York State Veterinary College. Annual conference for veterinarians. New York State Veterinary College, Cornell University, Ithaca, N. Y., Jan. 6-8, 1954. W. A. Hagan, dean.

Tennessee Veterinary Medical Association. Annual meeting. Nashville, Tenn., Jan. 11-12, 1954. H. W. Hayes, 734 Broadway, Knoxville, Tenn., secretary.

Indiana Veterinary Medical Association. Annual meeting. Hotel Severin, Indianapolis, Ind., Jan. 13-14, 1954. W. W. Garverick, Zionsville, Ind., secretary.

Kansas State Veterinary Medical Association. Annual meeting. Broadview Hotel, Wichita, Kan., Jan. 18-20, 1954, K. Maynard Curts, Kansas City, Kan., secretary.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Des Moines, Iowa, Jan. 19-21, 1954. F. B. Young, Waukee, Iowa, executive secretary.

Texas Veterinary Medical Association. Annual meeting. Hotel Galvez, Galveston, Texas, Jan. 24-26, 1954. Alvin A. Price, College Station, Texas, executive secretary.

California State Veterinary Medical Association. Midwinter convention. Veterinary Science Building, University of California, Davis, Calif., Jan. 25-27, 1954. Mr. Charles S. Travers, 3004 16th St., Room 208, San Francisco, Calif., executive secretary.

Minnesota State Veterinary Medical Society. Annual meeting. St. Paul Hotel, St. Paul, Minn., Jan. 25-27, 1954. B. S. Pomeroy, St. Paul, Minn., secretary.

North Carolina Veterinary Conference. North Carolina State College, Raleigh, N. Car., Jan. 26-28, 1954. C. D. Grinnells, chairman, veterinary conference.

Louisiana State University. Annual conference for veterinarians. Louisiana State University, Baton Rouge, La., Feb. 2-3, 1954. W. T. Oglesby, head, Department of Veterinary Science.

Illinois State Veterinary Medical Association. Annual meeting. Morrison Hotel, Chicago, Ill., Feb. 10-12, 1954. A. G. Misener, 6448 N. Clark St., Chicago 26, Ill., secretary.

(*Continued on p. 30*)

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(COMING MEETINGS—*continued from p. 28*)

Regularly Scheduled Meetings

Bay Counties Veterinary Medical Association, the second Tuesday of each month. David E. Madsen, 44 South 4th St., San Jose, Calif., secretary.

Cedar Valley Veterinary Association, the second Monday of each month (except July and August) at Black's Tea Room, Waterloo. F. E. Brutsman, Traer, Iowa, secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. W. E. Smith, 516 Oatman, Sanger, Calif., secretary.

Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel in Greensboro. Mr. Earl D. Adams, Greensboro, N. Car., secretary.

Chicago Veterinary Medical Association, the second Tuesday of each month. Robert C. Glover, 1021 Davis St., Evanston, Ill., secretary.

Coastal Bend Veterinary Association (Texas), the second Wednesday of each month. J. E. Hoban, 4301 S. Port Ave., Corpus Christi, Texas, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. J. R. Rosdail, Pomeroy, Iowa, secretary.

Cuyahoga County (Cleveland, Ohio) Veterinary Medical Association, the first Wednesday of each month—September through May (except January)—at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Roger W. Grundish, 4217 Mayfield Road, South Euclid 21, Ohio, secretary.

East Bay Veterinary Medical Association, bimonthly, the fourth Wednesday. Robert Clemens, 23352 Orchard, Hayward, Calif., secretary.

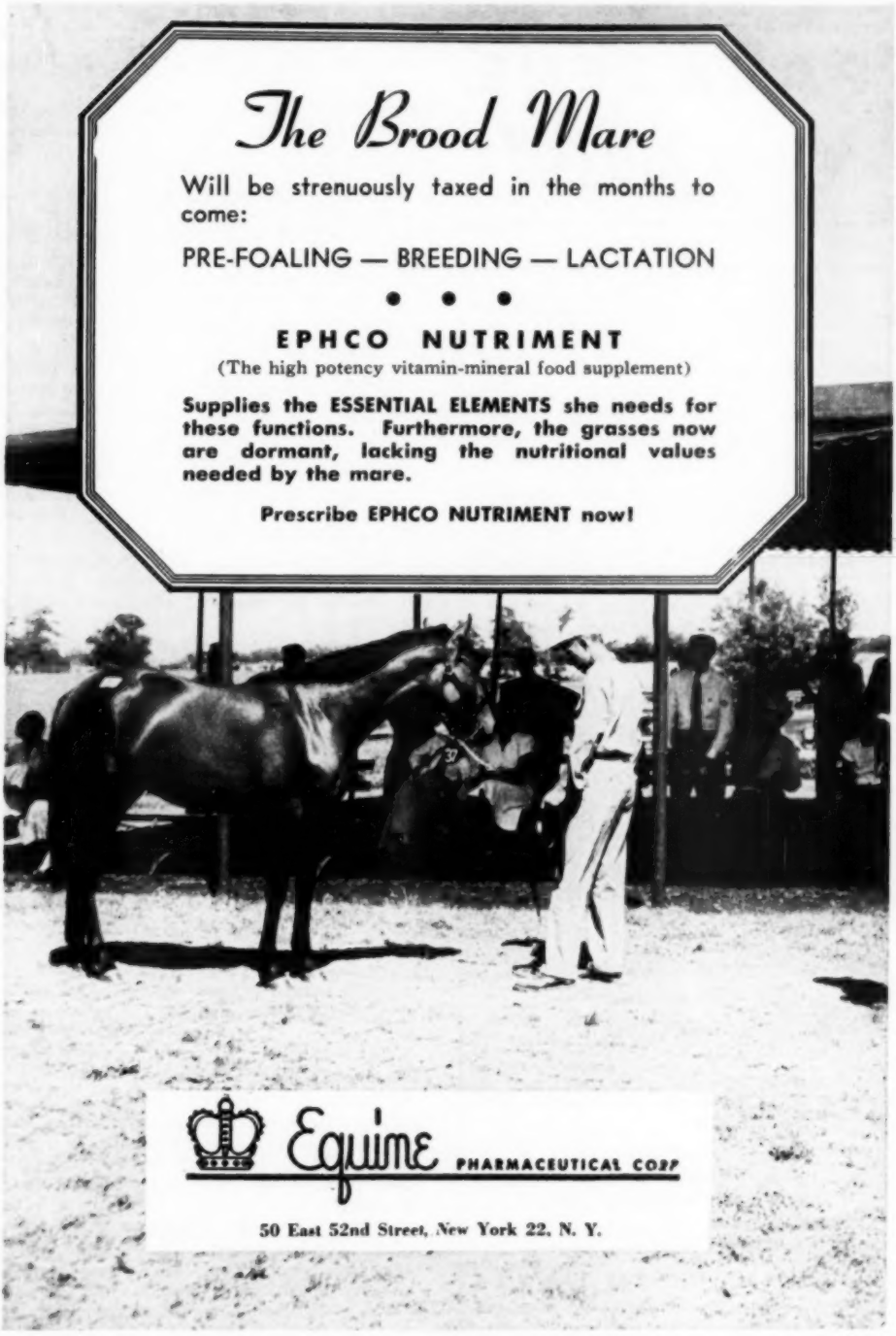
Fayette County Veterinary Association, Iowa, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Florida, North-East Florida Veterinary Medical Association, the second Thursday of each month, time and place specified monthly. J. O. Whiddon, 829 San Marco Blvd., Jacksonville, Fla.

Greater St. Louis Veterinary Medical Association, the first Friday of the month at the York Hotel, Sixth and Market Streets. Luther E. Fredrickson, Room 11, Municipal Courts Bldg., St. Louis, Mo., secretary.

Houston Veterinary Medical Association, Houston, Texas, the first Thursday of each month.

(*Continued on p. 32*)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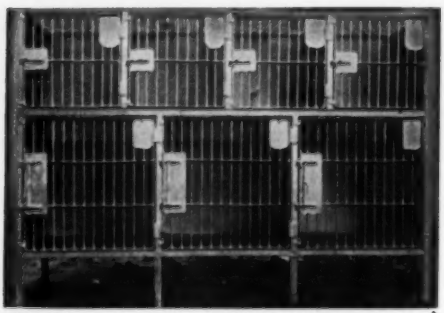

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(COMING MEETINGS—*continued from p. 30*)

Edward Lepon, Houston, Texas, secretary-treasurer.

Illinois Valley Veterinary Medical Association, the second Sunday evening of even-numbered months at the Jefferson Hotel, Peoria, Ill. S. M. McCully, Lacon, Ill., secretary.

Indiana Tenth District Veterinary Medical Association, third Thursday of each month. L. A. Snider, New Palestine, Ind., secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month, in Louisville or within a radius of 50 miles. E. M. Lang, 716 E. Broadway, Louisville, Ky., secretary.

Kansas City Small Animal Hospital Association, the first Monday of each month, at the Hotel Continental. T. M. Eagle, Parkville, Route 2, Mo., secretary.

Kansas City Veterinary Medical Association, the third Tuesday of each month, in the Hotel Continental, 11th and Baltimore, Kansas City, Mo. K. M. Curts, 70 Central Ave., Kansas City 18, Kan., secretary.

Kern County Veterinary Medical Association, the first Thursday of each month. Richard A. Stiern, 17 Niles St., Bakersfield, Calif., secretary.

Keystone Veterinary Medical Association, the Philadelphia County Medical Society Building, 301 S. 21st Street, Philadelphia, Pa., on the fourth Wednesday of each month. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

Kyowva Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Karl Mayer, 1531 Fourth Ave., Huntington, W. Va., secretary.

Maricopa County Veterinary Association, the second Tuesday of each month. Charles J. Prchal, 1722 East Almeria Road, Phoenix, Ariz., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from September through May, at the Academy of Medicine of Northern New Jersey, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Millburn Ave., Maplewood, N. J., secretary.

Michiana Veterinary Medical Association, the second Thursday of each month, at Hotel LaSalle, South Bend, Ind. Bruce Hostrawser, 2621 Mishawaka Ave., South Bend, Ind., secretary.

Michigan, Southeastern Veterinary Medical Society. Herman Kiefer Hospital, Detroit, Mich., the second Wednesday of each month from October through May.

(*Continued on p. 34*)

Five Famous Supplement Feeds

Help Correct Nutritional Deficiencies

Via-D-Mineral — 64,000 USP units vitamin D_2 per lb. Generally fed to dairy cattle but valuable to all 4-footed animals.

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(COMING MEETINGS—*continued from p. 32*)

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. George McCollister, 2146 Broad St., San Luis Obispo, Calif., secretary.

Milwaukee Veterinary Medical Association. Wisconsin Humane Society, 4150 N. Humbolt Ave., Milwaukee, Wis., the third Tuesday of each month. Kenneth G. Nicholson, 2161 N. Farwell Ave., Milwaukee, Wis., secretary.

Mobile-Baldwin Veterinary Medical Association, the first Tuesday of each month at the Hotel Admiral Simmes, Mobile, Ala. C. Eric Kennedy, Mobile, Ala., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 66 Marion Ave., Salinas, Calif., secretary.

New Castle County Veterinary Society, the second Wednesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. Harold Roberts, Paper Mill Road, Newark R3, Del., secretary.

New York City, Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63 St., New York City. C. R. Schroeder, Lederle Laboratories, Inc., Pearl River, N. Y., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday evening from September through June, at the Casa Mana Restaurant, Cedar Lane, Teaneck, N. J. Robert R. Shomer, 1680 Teaneck Road, N. J., secretary.

Northern San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month. Tom Hagan, Gen. Del., Escalon, Calif., secretary.

Oklahoma County Veterinary Medical Association, the second Wednesday of every month except July and August. W. C. Schilb, 4312 N. W. 23rd St., Oklahoma City, Okla., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month at 7:00 p.m. in the Antlers Hotel, San Bernardino, Calif. R. E. Hoadley, Coachella, Calif., secretary.

Orange County Veterinary Medical Association, bi-monthly. Donald E. Lind, 2643 N. Main, Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. P. H. Hand, Box 1035, Millbrae, Calif., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month at 7:00 p.m. in Mull's Motel in Hickory, N. Car. G. V. McCranie, Hickory, N. Car., secretary.

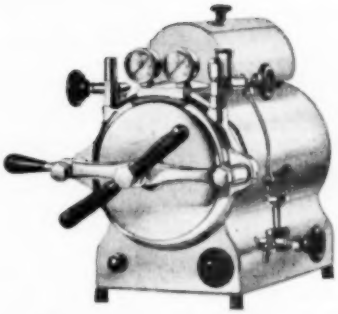
Pima County (Arizona) Veterinary Medical Association, the third Wednesday of each month.

(*Continued on p. 36*)

MOIST HEAT?
DRY HEAT?

You get both *only* with

PELTON AUTOCLAVES



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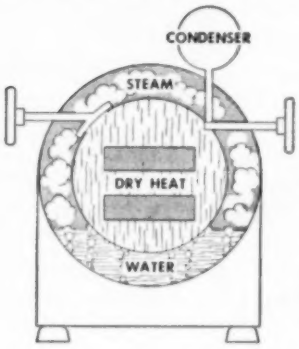

You need DRY HEAT to sterilize instruments that corrode readily

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Name_____

Address_____

City, State_____

(COMING MEETINGS—*continued from p. 34*)

in Tucson. R. W. Adami, 2103 S. 6th Ave., Tucson, Ariz., resident secretary.

Portland (Oregon) Veterinary Medical Association, the second Tuesday of each month, in the Auditorium of the Upjohn Company. Victor T. Oliver, 9705 S.W. Barbur Blvd., Portland 19, Ore., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. H. M. Strandberg, 203 D St., Petaluma, Calif., secretary.

Roanoke-Tar (N. Car.) Veterinary Medical Association, the first Friday of each month, time and place specified monthly. C. B. Randall, Kinston, N. Car., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. S. M. Foster, 430 College, Woodland, Calif., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. F. Ferguson, 1702 S. Dort Highway, Flint, Mich., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. Warren J. Dedrick, 904 S. Lemon, El Cajon, Calif., secretary.

Santa Barbara-Ventura Counties Veterinary Medical Association, Friday evenings every sixth week. Dee Wodars McDermott, 5879 Hollister, Coleta, Calif., secretary.

Southern California Veterinary Medical Association, the third Wednesday of each month. R. W. Sprowl, 11756 San Vicente Blvd., Los Angeles 49, Calif., secretary.

South Florida Veterinary Society, the third Tuesday of each month, at the Seven Seas Restaurant, Miami, Fla. E. A. Majilton, 1093 N. E. 79th St., Miami, Fla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month, in Director's Parlor of the Brookside State Bank, Tulsa, Okla. John Carnes, Muskogee, Okla., secretary.

Foreign Meetings

Second Pan American Congress of Veterinary Medicine. Sao Paulo, Brazil, April 3-10, 1954. Dr. Joao Soares Veiga, chairman; Dr. Virginia Buff D'Apice, secretary general, P.O. Box 7064, Sao Paulo, Brazil.

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burns	metritis
ulcers	retained placenta
fistulas	otitis
gangrene	pink eye
eczema	bovine mastitis

porcine necrotic enteritis

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Topical

Furacin Solution Veterinary 0.2%—rubber stoppered bottles of 500 cc., for use in mastitis. Also 1 pt. and 1 gal. bottles with screw caps.

Furacin Dressing Veterinary 0.2%—4 oz. and 1 lb. jars.

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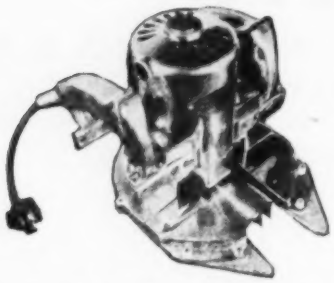
A Sweeping Advance in Dehorning Cattle



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UNIVERSAL HORN SAW removes horns easily when growing at improper angles
UNIVERSAL HORN SAW lessens pain and shock to animals
UNIVERSAL HORN SAW eliminates possibility of splintering the horn base
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YES! UNIVERSAL HORN SAW is the answer for fast, safe, easy method of dehorning cattle.

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Veterinarian wanted for institutional work; 40-hour week. Splendid opportunity for clinical small animal experience. New York state license essential. Address "Box P 2," c/o JOURNAL of the AVMA.

Veterinarian wanted, California license, for small animal practice in modern hospital in Los Angeles suburb. Excellent working conditions, salary, and percentage. Capable practitioner can net in excess of $10,000 annually. Inquiries confidential. Dr. C. H. Ozanian, Bellflower Veterinary Hospital, Bellflower, Calif.

(*Continued on p. 40*)

STRONTIUM MEDICAL APPLICATOR
. . . for Beta Ray Therapy

Tracerlab's unique Strontium Medical Applicator is a simple, compact instrument which allows *beta* rays to be used in the treatment of such eye conditions as corneal inflamatory lesions of dogs and superficial ocular neoplasms of horses and cows.*

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The standard RA-1A Strontium Applicator contains a forty millicurie source, but may be ordered with strengths of up to one hundred millicuries, upon approval of the United States Atomic Energy Commission.

We will be happy to send a copy of *"Beta Ray Therapy in Ocular Diseases of Animals"** on request and to supply appropriate A.E.C. forms with instructions for completing them.

* *Beta Ray Therapy in Ocular Diseases of Animals,* Catcott, et al; Journal A.V.M.A., March, 1953; p. 172-175.

Photos - Jrnl. A.V.M.A., March '53

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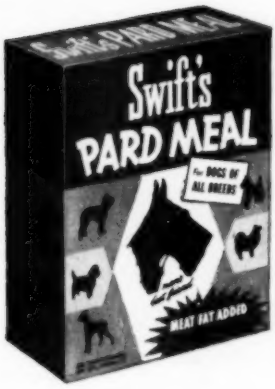

One time when your experience tells you nothing...

EVERY once in a while that tried-and-true yardstick, *experience*, gets nudged aside.

Here is a case in point. Your experience tells you that it's a rare dog who really *likes* a dry-type food. Your experience tells you that even the best of dry dog foods have missed on "appetite appeal." Economical, yes. Nutritious, often. But tasty, never.

Experience tells you to be *skeptical* about a dry dog food that claims to have a "flavor dogs can't resist."

With Swift's Pard Meal, your experience tells you nothing. Be skeptical if you like. Doubt as you please. But try a bag.

Swift's Pard Meal is the new dry-type dog food that actually has a FLAVOR DOGS CAN'T RESIST. It's been tested and tested and proven. It's the dog meal with an appetite appeal such as you didn't believe there'd ever be.

Swift's Pard Meal contains extra amounts of rich meat fat for super energy and fuller nutrition. It's a kernel-type food, and homogenized of course. It won't flake or powder, and won't turn rancid. It's a complete diet—a dog needs nothing more.

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PARD is Swift's famous canned dog food. Here is the "pioneer" product of them all —for 20 years leading the way to today's high level of nutritional excellence in commercial dog foods. A dog needs nothing more—you can recommend nothing better than Pard.

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100,000 units penicillin

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100,000 units penicillin
50,000 mcg. dihydrostreptomycin

for control of Mastitis

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package of 100	13.00	19.00
package of 500	11.00*	19.00*

(*per hundred)

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package of 25	$4.00	$6.00
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*ADDRESS*_____

*CITY*_____ *STATE*_____

(CLASSIFIED ADS—*continued from p. 38*)

Veterinarian wanted for Southern California small animal hospital. Applicant must have California license and be capable of managing hospital. State experience and give full details. Address Leroy E. Schafer, D.V.M., 5536 Vineland Avenue, North Hollywood, California.

Experienced graduate veterinarian wanted for mixed animal practice in Middlewest state. Must have own car. Send qualifications and salary expected in first letter. Address "Box P 11," c/o JOURNAL of the AVMA.

Veterinarian wanted for mixed practice in Central New York State. Good starting salary; opportunity for partnership in near future. Good living quarters. Address "Box P 14," c/o JOURNAL of the AVMA.

Exceptional opportunity for licensed veterinarian in small animal hospital in large town in New England. Address "Box P 18," c/o JOURNAL of the AVMA.

Remittance must accompany advertisement

Veterinarian wanted, experienced in small animal diagnosis and surgery. Must be licensed in Ohio. Starting salary $125 per week, bonus, and opportunity to buy into practice. Address Dr. Powell's Memorial Pet Hospital Inc., 10608 Carnegie Ave., Cleveland 6, Ohio.

(*Continued on p. 42*)

NOW available! Metal covered cases for your serums and instruments!

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precision veterinary equipment

CONSTANT RESEARCH
means Better Products at
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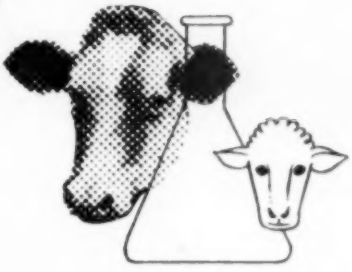

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Proven by laboratory analysis and field test to be the most potent, most effective rumen inoculant and stimulant yet developed, RU-ZYME contains 2 billion viable rumen bacteria per gram . . . *PLUS* mold enzyme cultures which contain unidentified growth factors that accelerate natural rumen function. Improved scientific production methods now enable us to pass on to our customers heavy price reductions, as follows:

NEW LOWER PRICE — **Only $10.50 per case of twelve 75-gram jars; 1-lb. canisters $4.50**

2. RU-BOLS, a new companion product to RU-ZYME in Bolus Form

RU-BOLS (a 480-grain bolus) contain Ru-Zyme . . . with a guarantee of 45 billion viable anaerobic rumen bacteria per bolus. Ru-Bols are packed 25 to the carton and are priced at $7.00 per carton.

If you want assured results at minimum cost . . . USE RU-ZYME or RU-BOLS

RU-ZYME and RU-BOLS are available from your Veterinary Supply Dealer, or write us direct. ►

THE RUMELK COMPANY, Inc. Salem Virginia

Provides an accurate pattern against which to cut with knife or razor blade. Fits firmly, cannot move or slip when clamped into position. Made of non-rusting, light, cast aluminum, highly polished. Lasts a lifetime with minimum care. Simplicity of design and construction reduces possibility of breakage or mechanical failure. Forms immediately available to provide distinctive marking of these breeds:

Boxer — postpaid $15.00
Boston Terrier — postpaid $15.00
Great Dane — postpaid $15.00
Doberman — postpaid $15.00
Set of above four — postpaid $50.00

These patented "championship" forms are patterned after markings of winners of top honors in show competition. Forms for other breeds made on special order. Sold to veterinarians only. Send check or money order.

MacALLAN LABORATORIES
Route No. 2, Box 420 Lansing, Michigan

SULFADAC Curts

Sulfamerazine 3%

Sulfapyridine 4%

Sulfathiazole 6%

(13% total sulfonamide—2 grains per cc.)

The Original Triple Sulfa Parenteral Solution for Veterinary Use

Still the best sulfa combination for the many infections encountered in general practice.

250cc	1.65	6-250cc	9.00
500cc	3.00	6-500cc	16.80

(larger quantity prices on request)

CURTS-FOLSE LABORATORIES

Pharmaceutical Manufacturers to the Veterinary Profession since 1918

73 Central Avenue Kansas City, Kansas

(CLASSIFIED ADS—*continued from p. 40*)

Wanted—Positions

Woman, graduate approved school, small animal experience, seeks position in small animal or mixed practice leading to partnership or purchase. Address "Box P 4" c/o JOURNAL of the AVMA.

Graduate veterinarian, approved school 1944, desires assistantship, partnership, or purchase practice. Serving in Air Force under doctor draft; available Feb. 1, 1954. Experienced in large and small animals. References; married, 31 years old. Licensed in Colorado, Oregon, Nebraska but will locate anywhere. Address "Box K 7," c/o JOURNAL of the AVMA.

Licensed veterinarian, age 34, married, experience in large and small animal practice, desires position with practitioner in Connecticut or Massachusetts. Address "Box P 12," c/o JOURNAL of the AVMA.

European veterinarian, recognized school, middle aged, married, no children, looking for permanent employment in a mixed or small animal practice. Address "Box P 16," c/o JOURNAL of the AVMA.

Association wanted with small animal hospital which offers future for young, experienced veterinarian. Graduate of AVMA-approved school. Available immediately. Married. Address "Box P 23," c/o JOURNAL of the AVMA.

Veterinarian seeks permanent position in small animal or mixed practice. Prefer Ohio or Indiana. Licensed in Ohio; 5 years' experience in small animal practice. Address "Box P 24," c/o JOURNAL of the AVMA.

Experienced veterinarian desires assistantship or partnership in an established small or mixed practice. Licensed in Arkansas, Missouri, and South Dakota but will locate anywhere. Address "Box P 19," c/o JOURNAL of the AVMA.

Wanted—Practices

Small animal practice and hospital wanted. Veterinarian, 38 years of age, with 13 years' experience, mostly large animal, wishes to make a change and purchase a modern, going, progressive, and prosperous small animal hospital with high class clientele. Must be an established hospital. Prefer a practice and hospital large enough for two veterinarians; 60 kennels or better. Send complete information, including photographs of hospital, real estate value, equipment, competition, annual gross, number of cages, outside runs, parking facilities for clients, and population of town. Substantial cash available. Would consider purchase of 50% of small animal hospital as described above with right man. Address "Box P 10," c/o JOURNAL of the AVMA.

(*Continued on p. 46*)

40 years of service to the veterinary field

GOSHEN LABORATORIES, INC.
GOSHEN, NEW YORK
"THE PRACTITIONER'S HOUSE"

When you buy, buy from an ethical house

quick "pick-up"

for respiratory diseases in swine

AMMOCHROME POWDER

Easily Soluble • Palatable to Swine

The improved and economical method of dispensing guaiacol. Just add recommended quantities of NLC AMMOCHROME POWDER to the drinking water of infected pigs, respiratory congestion is eased and animals show a quick "pick-up."

1 lb. dissolved in 1 gal. of water makes a solution suitable for treating the same type of infections as are benefited by the use of a liquid "tasteless" guaiacol compound. Costs 40% less.

COMPOSITION: 19% Potassium Guaiacol Sulphonate with Potassium and Ammonium Salts in a special aromatic base. Indicated in disease of the upper respiratory tract, pneumonia, bronchitis, laryngitis, swine flu.

Available in 1 lb. containers featuring double label for dispensing purposes.

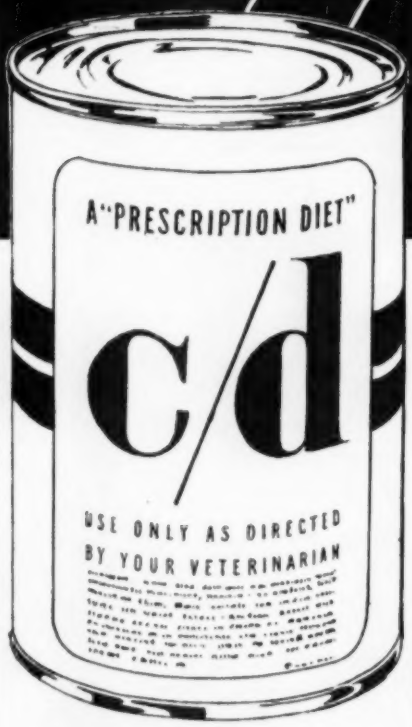
NLC

SERVING GRADUATE VETERINARIANS EXCLUSIVELY

THE NATIONAL LABORATORIES CORPORATION

KANSAS CITY

Introducing
.. A New Member
of the
Diet Family



This new product for cats joins the Hill family of Prescription Diets which includes:

k/d — for Nephritic Conditions
r/d — for Obesity Correction
p/d — for Reproduction & Lactation
i/d — for Intestional Disorders

Available only to Graduate Veterinarians

Write us for complete information

HILL PACKING COMPANY

P.O. Box 148 Topeka, Kansas

Correspondence

Following the award of the Twelfth International Veterinary Congress Prize to Dr. L. Van Es at the Ninetieth Annual A.V.M.A. meeting in Toronto last July, Dr. Van Es wrote:

Lincoln, Neb., Aug. 24, 1953

Dear Dr. Hardenbergh:

Will you kindly accept and convey to our fellow members this expression of my appreciation of the honor which they so generously bestowed upon me.

With my best wishes, I remain

Sincerely yours,
s/ L. Van Es

Dr. F. E. Eads Promoted

Dr. F. E. Eads, Birmingham, Mich., has been promoted to manager of the department of veterinary medicine at Parke, Davis & Company.

Dr. Eads, assistant manager of the department since April 1, 1951, succeeds the late Dr. Hilton O. von Rosenberg, who died July 25 after an illness of more than a year.

Dr. F. E. Eads

Born at Elnora, Ind., Dr. Eads spent most of his early years in Urbana, Ill., where he took premedical courses at the University of Illinois. He was graduated from Michigan State College in 1941 with the degree of doctor of veterinary medicine.

After working for the federal government in Wisconsin a few months, Dr. Eads joined the Illinois Department of Agriculture. In 1946, he returned to Michigan State as assistant professor and completed his master's degree in surgery and medicine in 1949.

Your Office is Always as Near as Your Car with Motorola 2-way Radio

Now, you can speed rounds—handle up to 30% more calls daily—with easy-to-operate Motorola Radio

Instant contact with home or office is yours *at all times* with Motorola 2-way Radio in your car. You save time, mileage and effort, yet increase calls and improve service to your clients. Motorola radio lets you set up schedules *en route* . . . helps start treatments faster. When necessary, you radio emergency first aid to clients via your office.

You get lightning-fast communications from the moment experienced Motorola engineers supervise the installation of your set. No tests to pass—no technical skill needed. Eight exclusive Motorola features assure superior performance from the very start. Years-ahead Motorola engineering guarantees time-tested durability.

For Details—Write Dept. 2286-VM Today!

NO TECHNICAL SKILL NEEDED

Just Push a Button... and YOU'RE ON THE AIR!

national **Motorola** RADIO COMMUNICATIONS *service*

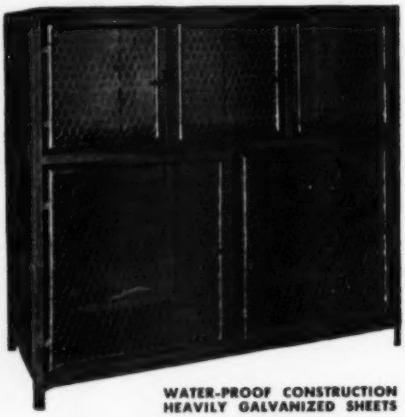
Motorola Communications & Electronics, Inc.
A SUBSIDIARY OF MOTOROLA, INC.
900 N. Kilbourn Ave., Chicago 51, Illinois • Rogers Majestic Electronics Ltd., Toronto, Canada

WHEN ANSWERING THIS ADVERTISEMENT, PLEASE MENTION "ITEM 5372"

"Water-Proof"

5 COMPARTMENT STALLS

$179.90



WATER-PROOF CONSTRUCTION
HEAVILY GALVANIZED SHEETS

NO EXTRA CHARGE!

Bottoms are water-proof trays with ½" turned up edges, heavily soldered together. Braced, aluminum painted. 1½" angle iron frames. Door frames 1" O. D. pipe. Dog proof mesh filler welded to frame. All sheets heavily galvanized.

Completely Assembled. Satisfaction Guaranteed.
SIZES: 3 upper stalls 24" x 24" x 28" deep.
2 lower stalls 36" x 36" x 28" deep.
Overall size: 6' wide x 5'6" high x 28½" deep.
Stalls Stand 6 in. off Floor.

SAFETY

KENNEL RUNS

The low cost will surprise you!

Ford DOUBLE FRAME Panel Runs insure SAFETY for your dogs. Chain link fabric is rust resistant, cannot be spread; permanently locked by INNER BAR FRAME. NO TIE WIRES TO RUST. Clamp together. No bolt holes to match. Portable or permanent construction.

Made in sizes to fit your requirements. 4, 5, and 6 ft. heights. Lengths 2 to 14 ft. panels.

WRITE FOR LITERATURE AND PRICES

Kennel Runs — Pup Pens — Stalls and Cages

Ornamental and Miscellaneous Iron.

FORD FENCE CO., Inc.

6542 Westfield Boulevard

INDIANAPOLIS 20, INDIANA

(CLASSIFIED ADS—*continued from p. 42*)

Graduate of approved school, desires to buy or lease large animal practice, or large animal part of a mixed practice. Single; over two years' experience in mixed practice. Licensed in Pacific Coast, and North Central states. Address "Box P 13," c/o JOURNAL of the AVMA.

Practice wanted in New Jersey, Pennsylvania, or Delaware; preferably small animal. Substantial down payment available. Will consider partnership with right man. Expect release soon from Army. Address "Box P 25," c/o JOURNAL of the AVMA.

Experienced veterinarian desires purchase active small animal practice, with residence preferred. Licensed in New York, Pennsylvania, Ohio, Maryland, Connecticut, Massachusetts. Substantial cash for right proposition. Address "Box P 26," c/o JOURNAL of the AVMA.

For Sale or Lease—Practices

Small animal hospital for sale or lease, with option to buy. Florida, lower east coast. Information on request. Address "Box P 3," c/o JOURNAL of the AVMA.

Due to business interests in another city, the owner of the Animal Hospital of Brooklyn, 1793 Nostrand Avenue, Brooklyn, N.Y., wishes to sell his practice there. The hospital was recently renovated; has 50 cages, good lease, outside runs, isolation wards and all modern equipment, including x-ray. This practice can be purchased for this years' gross of $25,000.

(*Continued on p. 48*)

Sold only to Graduate Veterinarians

FUNGASARC

for the effective treatment of skin conditions

Destroys fungi; sarcoptes scabei canis; demodex canis; mites; fleas; lice. Repels ticks. Non Staining; not greasy; has no objectionable odor, destroys odors of external origin. Non Toxic; may be used daily in recommended dilution. Concentrated; one gallon makes four.

Gallon **$13.95** **Makes 4 gallons**

Quart **$4.00** **Makes a gallon**

Available nationally through well known Distributors

Osco Chemical Company, Inc.

P.O. Box 2157, Atlanta 1, Georgia

greater versatility

because of no pressor effects

P.O.P.
purified oxytocic principle

P.O.P. (Armour) secures its specific effects on the uterus and mammary glands without the pressor effects typical of unpurified or only partially purified posterior pituitary preparations. P.O.P. (Armour) minimizes in small animals the vomiting effect that less purified preparations impart.

Double action—Plus economy
P.O.P. (Armour) causes rapid and powerful uterine contractions of a rhythmic nature and exhibits milk-ejecting activity ("milk letdown"). Economy of treatment is another distinct advantage of P.O.P. (Armour). It contains 20 U.S.P. units of oxytocin, and less than 0.4 units of pressor activity per cc.—double U.S.P. strength.

Indications
Obstetrics (prepartum and postpartum); uterine hemorrhage; pyometra and chronic endometritis; lactation; stimulation of "milk letdown." A dramatic effect has been shown recently in the treatment of mastitis.

How supplied
In 5 cc. and 10 cc. multiple dose vials, 20 U.S.P. Units per cc.

*Armour's trade name for veterinary purified oxytocic principle preparation.

Sold to Graduate Veterinarians Only

VETERINARY ARMOUR LABORATORIES

A Division of Armour and Company • Kansas City 18, Kansas

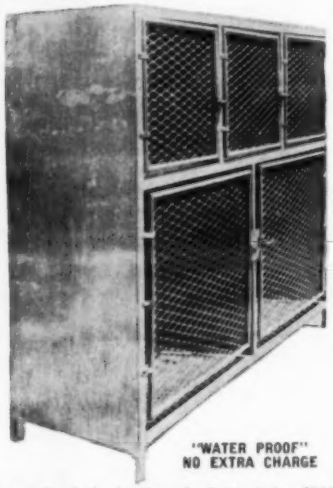
Practice, primarily dairy, for sale in San Joaquin Valley of California. Hospital, drugs, equipment, including 2-way radio. Gross $35,000 year; price $25,000. Terms can be arranged. Address "Box P 5," c/o JOURNAL of the AVMA.

A top, mixed pratice like this is seldom for sale. In the very best hog and cattle-feeding area in the Middlewest. Practice grosses from $50,000 to $75,000; established 30 years. Hospital and home worth asking price. Practice should net selling price first year; 2-way radio. $25,000 cash. Address "Box P 6," c/o JOURNAL of the AVMA.

Retiring veterinarian wishes to sell equipment and lease or sell hospital. Practice established 1919; only hospital zoned in city. Will help right man get established. Address Dr. George Adamson, 529 So. Crockett St., Sherman, Texas.

Hospital, 2-bedroom house, plus 15-acres land for sale. Small down payment. Good location for mixed practice. Would lease just the hospital and equipment. Address Dr. J. L. Davidson, The Upjohn Co., Kalamazoo, Mich.

5 COMPARTMENT KENNELS $169.50



"WATER PROOF" NO EXTRA CHARGE

Water-proof, sheets heavily galvanized, steel. 1½" angle steel welded frames. Doors—9 ga. 1½" steel mesh with steel angle frame. Assembled complete. 5' 6" high. 6 ft. long, 6 in. off ground. Painted. Serving leading Universities.

Compare before you buy and you will agree that our kennel is excellently constructed, more costlier materials used, cheaper in price and you actually save from $25.00 to $75.00 for the same kennel constructed elsewhere. All floors are flush for easy cleaning. Over 3,000 satisfied customers. Heavy duty casters, $12.50 extra. Sorry, no C.O.D.'s. Send check or M.O.

Write For Free 16-Page Catalog.

DEE-CHICAGO MFG. CO

Mfrs. of Dog Crates, Equipt. & Supplies Since 1912

7600 S. Campbell Ave., RE 7-3300, Chicago 29, Ill.

Death makes available for lease or sale animal hospital in West Virginia city of 50,000. Large animal, 40%; kennels for 50 small animals. Annual gross approximately $30,000. Address "Box P 9," c/o JOURNAL of the AVMA.

For sale, due to physical condition, the most modern practice in Baltimore, city of over a million population. Practice gross, over $30,000 per annum. Located in city; modern apartment over hospital. Hospital fully airconditioned, 75 kennels, automatic dog drier, x-ray, large drug supply, full instrumentation, modern design office with building. Sacrifice below cost at only $30,000 for everything. Must sell. Dr. E. I. Neserke, 1818 E. Fayette St., Baltimore 31, Md.

Small animal hospital near Los Angeles, for sale. No real estate; 40-animal capacity. Gross $26,000; $12,500 cash or $13,500 terms with $5,000 minimum down. Address "Box W 17," c/o JOURNAL of the AVMA.

Long-established practice for sale. Completely equipped veterinary hospital and boarding facilities located at present address since 1927. Retiring from business; exceptional opportunity at an interesting price. Address Clarence E. Shaw, 287 Flatbush Ave., Brooklyn 17, N.Y.

Fully equipped small animal practice for sale in Southern California. No real estate. Have other commitments so is priced to sell. Address "Box P 17," c/o JOURNAL of the AVMA.

(*Continued on p. 50*)

Feed for Top Condition at Low Cost!

When you feed *nothing* but Kasco Complete Dog Food and water—(no extra meat or supplements of any kind)—you're sure of two results:

1. Top-notch condition
2. Low feeding costs

Kasco is a dog-licious diet, nutritionally complete—so economical it can't be beat!

KASCO MILLS, INC., TOLEDO, OHIO - WAVERLY, N.Y.

NOW! Fast . . . Safe . . . SURE Way To Dehorn Calves

Sunbeam STEWART

AUTOMATIC HEAT CONTROL CALF DEHORNER

Electric dehorning with the new Sunbeam Stewart Calf Dehorner is *fast, safe* and *certain.* Dehorning can be done any time of year. Only a few minutes are required for the entire job. No loss of blood—no open flesh wound—no bad after effects. Dehorner has automatic control for steady high heat for dehorning or soldering. Cool, easy-grip handle. Complete with heavy duty soldering tip. $15.95.

- Automatic Control for Steady Heat
- Fast—Easy to Use
- No Loss of Blood—No Open Flesh Wound

Becomes heavy-duty soldering iron when soldering tip is attached.

Write for information about Sunbeam Stewart Dehorner and animal clipping equipment

Sunbeam CORPORATION *(formerly Chicago Flexible Shaft Company)*

5600 W. Roosevelt Rd., Dept. 47, Chicago 50, Illinois

versatile . . .

economical

medium size

SERVALL

Angle Centrifuges

Research Scientists and Technicians everywhere acclaim the Medium Size SERVALL Angle Centrifuges: Type M, Type SP and Type SP/X. All employ the same motor (which is equipped with an exclusive self-centering device and dynamical balance) and all are adaptable to various tube sizes by means of simple, convenient adapters.

Type M, SP and **SP/X Servall** Angle Centrifuges have a rim dia. of 10 in., speed of 5000 r.p.m. and hold tubes at a 40° angle.

Type M holds 27 round bottom tubes of maximum 15cc and is widely used in hospital routine work. Has 27 metal holders.

Type SP, frequently specified for routine or research work, holds six 75cc plain or 70cc constricted neck tubes. (Special unbreakable cellulose nitrate tubes of 80cc or less are also available.) With adapters it will accommodate larger numbers of small tubes.

Type SP/X holds ten 50cc and five 15cc tubes. Readily available adapters for smaller tubes provide utmost versatility.

Write for Bulletin VM 111

Ivan Sorvall, inc.

210 FIFTH AVENUE, NEW YORK 10, N. Y.

(CLASSIFIED ADS—*continued from p. 48*)

Modern newly constructed small animal hospital for sale; fully equipped, located in Cleveland, Ohio, close to one of the wealthiest suburbs. Present gross, $36,000; could do more with a conscientious assistant. Priced reasonably; will arrange terms for the right man. Reason for selling is difficulty in obtaining qualified help. Address Dr. Powell's Memorial Pet Hospital, Inc., 10608 Carnegie Ave., Cleveland 6, Ohio.

Outstanding small animal hospital for sale in Southern California; well-located, zone protected, excellent clientele, modern brick construction, emaculate, fully equipped, 60 cages. Health forces sale. Terms, $25,000 down. Address "Box P 15," c/o JOURNAL of the AVMA.

Mixed practice for sale in southwestern Oklahoma. Complete modern hospital, kennel, and large animal facilities. Only veterinarian in large territory; could support two men. Gross, $23,000; price, $9,000, $5,000 down, balance on terms. Address "Box M 1," c/o JOURNAL of the AVMA.

Mixed practice for sale or lease in 100,000 population area of Massachusetts. New 7-room home and small animal hospital. Modernly equipped with x-ray, steel cage units, complete drug inventory, etc. Buildings on 3-acre main route, corner. Immediately available. Reason for selling, health. Sale price, $29,000 complete. Will lease for $450 per month with option to buy. Address "Box P 7," c/o JOURNAL of the AVMA.

Mixed practice for sale; two-thirds small animal. Beautiful irrigated, recreational, desert valley, Southern California. Young practice, low price, bright future of continual growth and expansion. Price includes reasonably equipped hospital; 2-bedroom furnished home connected. Address "Box P 8," c/o JOURNAL of the AVMA.

Remittance must accompany advertisement

Well-established large animal practice for sale in Pacific Northwest; includes home, office, drugs, and equipment. Address "Box P 20," c/o JOURNAL of the AVMA.

Small animal hospital for lease. Established over 20 years; fully equipped. Superb location in rapidly expanding Southern California area. Excellent opportunity for experienced, ambitious veterinarian. Address "Box P 21," c/o JOURNAL of the AVMA.

Large animal practice for sale in Northern Illinois. Grossing $23,000; will sell for real estate value. Address "Box P 22," c/o JOURNAL of the AVMA.

Miscellaneous

For sale—Raytheon two-way radio (25-50 meg. band). 50-watt base station, 30-watt mobile unit. Complete except for base station antenna. Price $200. Address Box 225, Cambridge, Md.

Artificial insemination instruments. Disposable obstetrical sleeves 20¢ each. Package of 20 including detachable chest band—$5, postpaid. Samples and complete catalog on request. Breeders Equipment Co., Flourtown, Pa.

(*Continued on p. 52*)

Serving The Southern Veterinarian
For Over Twenty Years

L. A. MOSHER Company
Atlanta, Ga.

Albany, Ga. Louisville, Ky. Memphis, Tenn. Miami, Fla. Raleigh, N. C.

For TEAT PROBLEMS IN YOUR PRACTICE

SULFA impregnated—
surgical dressings for the Teat Canal

To maintain unrestricted milk flow and provide antiseptic protection are of first importance in the care of injured teats, scab teats, and in post operative therapy.

Dr. Naylor Dilators act as an internal bandage to the teat canal. They provide gentle, non-irritating support and broad spectrum germicidal activity to injured teat mucosa. They promote normal tissue repair with a minimum of altered milking function of the streak canal.

STERILIZED — MEDICATED

Dr. Naylor Dilators are sterilized and medicated prior to packaging. The medication is IN the Dilators and is released slowly to maintain a prolonged antiseptic level.

Dr. Naylor Dilators are soft, supportive surgical dressings impregnated with Sulfathiazole and Methyl Violet. Fit either large or small teats. Smooth, rounded tip for easy insertion.

Dr. Naylor's

MEDICATED TEAT DILATORS

DISPENSING PACKAGE (Contains 12 Dilators) 3.00 per doz. →

H. W. NAYLOR CO., Manufacturing Chemists, MORRIS, N.Y.

NEW IMPROVED RANCH RECORD VETERINARY SYRINGE

The Ranch Syringe was improved lately with a new two piece top cap. The plunger can be removed for sterilization without removing the glass barrel from the metal frame. This improvement makes it easier to sterilize and eliminates breakage. Smooth operation and accurate dosage is assured at all times.

The Ranch Syringe is made with finger bars or three rings.

Inquire at your nearest veterinary dealer or wholesaler about this new improved outstanding product.

Sizes 2cc. to 40cc.

Literature Upon Request

Boston Instrument Mfg. Co. Inc.,
50 Thayer St., Boston 18, Mass.

Pregnancy Diagnosis—in mare from 45th to 150th day. Write for vials and mailing tubes. Price: $7.00; 2 or more tests, $6.00 each. Pregnancy Diagnostic Laboratories, H. S. Lames, D.V.M., Dysart, Iowa.

Kennel blanks, boarding records, boarding contracts, pedigree blanks, stud certificates—anything in records. Write us. *Dog World*, $3, year; $5, two years. Judy Publishing Co., 3323 S. Michigan Blvd., Chicago 16.

BROKEN TEETH

—repaired in bottom clipper blades. Top and bottom blades sharpened to match. Save money—Guaranteed. Prices on Request

HIGHLY SPECIALIZED SHARPENING

Sales—Repairing on Oster and Stewart clippers.

Sharpened Blades Tested on Rabbit Fur

OSTER A-2 $.75 STEWART $1.00

Prompt Service—Est. 17 years

MAIL TO NEWFOUNDLAND 9-3412

CLIPPER SERVICE OAK RIDGE NEW JERSEY

Clipper Blade Sharpening

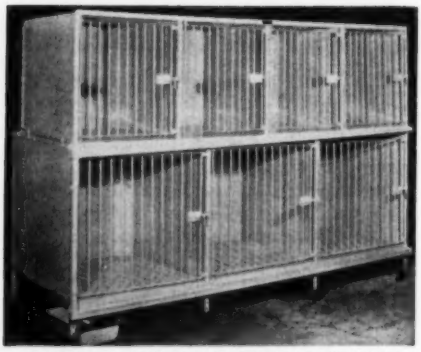
Have your clipper blades sharpened by an expert with 28 years of practical factory training. We know how your blades must be ground to cut like new. Also clipper repairing. All work guaranteed to satisfy you or money back. Now serving veterinary colleges, and over 500 satisfied veterinarians. Why not you? 24-hour service. We ship insured prepaid. Avoid C.O.D. charges; enclose 75¢ for each set with blades. We are headquarters for Oster and Stewart clippers and blades. Complete clipping supplies; upper and lower replacements. Write for information on Oster and Stewart quality clippers and products. Service Grinding and Supply Co., 903 Chicago St., Racine, Wis.

Bovine Prolapse Preventer—Payton Utero-Vaginal Prolapse Preventer. Quickly, easily applied to any size cow. Positive protection. Re-usable, non-irritating, sanitary. Noninterference with placenta release. Excellent for vaginal protrusion; dispensing. See article JOURNAL of the AVMA, December, 1951. Only $3.00; two for $5.00; six for $12.00. Prepaid. Dr. Jerome Payton, Morris, N.Y.

Medical Field Operating Lamps, new, portable, run on any current. Wilmot Castle, mfg. $40. FOB Orangeburg. Also boating supplies, oddities. Request catalogue. Sailors Surplus, Inc., Orangeburg 19, N.Y.

(*Continued on p. 54*)

DE LUXE CAGES AND DOG DRYER



IMPROVED 7-CAGE DE LUXE DRAIN UNIT

The small size is 7½ feet long with 3-30" cages in bottom and the large unit 9 feet long with 3-36" cages in bottom.

Also available in removable tray style, or plain flat floor style without gutter drains in front.

Our units are available in 3, 5, 7 or 12 cage units, or any combination you need.

Write for NEW cage booklet; also DRYER FOLDER

The 1953 price list is now available

Baltimore Wire & Iron Works

514 N. Jasper Street Baltimore 1, Maryland

Our improved dog dryer with entirely transparent doors and larger blower is proving most satisfactory to our numerous users. Many have recommended them to their friends saying "It's doing a wonderful job."

Better absorption and distribution with these combined sulfas

MERAMETH®

STERILE SOLUTION OF 5% SODIUM SULFAMERAZINE AND 5% SODIUM SULFAMETHAZINE

ACTIONS AND USES: Severe general infections in animals may be quickly and more safely controlled by intravenous or intraperitoneal injections of MERAMETH. Mastitis is promptly treated with MERAMETH and antibiotics (penicillin and dihydrostreptomycin sulfate) by direct intramammary infusion.

With MERAMETH you have: 1. Complete choice of injection route. 2. A *combination* of prolonged blood levels *and* high tissue concentrations. 3. Efficacy against a broader spectrum of gram-positive and gram-negative bacteria than with any sulfonamide alone. 4. Increased safety without interference to renal elimination.

Indications—In cattle: hemorrhagic septicemia, pneumonia, septicemia with metritis and mastitis, foot rot, calf scours, and calf diphtheria. In pigs: necrotic enteritis, hemorrhagic septicemia. In horses: shipping fever, equine influenza. In dogs and cats: secondary infection associated with distemper.

SUPPLIED: 250 cc. and 500 cc. rubber-capped bottles. *Sharp & Dohme, Veterinary Department.*

(CLASSIFIED ADS—*continued from p. 52*)

Received Too Late to Classify

High-type, energetic veterinarian wanted, preferably with some experience in dairy work. Must be from an accredited school and able to furnish references. Will give fine opportunity for right man, leading to future partnership. Address "Box P 27," c/o JOURNAL of the AVMA.

Experienced veterinarian, 1941 graduate, just separated from Army, desires to purchase or lease small animal practice. Licensed in Michigan, Maryland, Ohio, Texas, and Oklahoma. Can make substantial cash payment on a good going practice. Address "Box P 28," c/o JOURNAL of the AVMA.

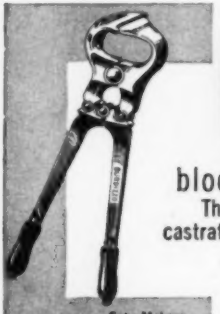
Kennel Cage Units

of Wire Mesh and Metal

IN TWO TIERS: 4, 6, and 8 cage units
IN THREE TIERS: 6, 9, and 12 cage units
Sanitary, durable, and easily maintained; galvanized materials; removable pans; hand woven wire mesh doors, maximum light and ventilation; sized for dogs and cats; removable partitions; shipped crated; easily assembled.

THE EIGHT CAGE UNIT
manufacturers

EAST RIVER WIRE WORKS
39-40 Twenty-first St., Long Is. City 1, N.Y.



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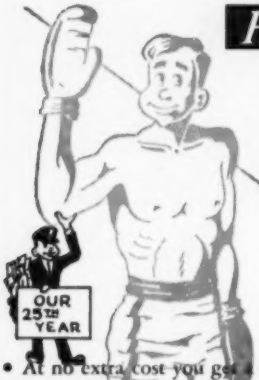

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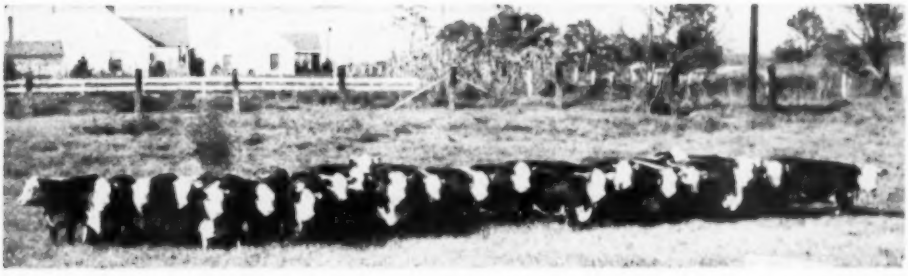

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